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Symposium on Snake Bite.

THE PRESENT POSITION OF SNAKE BITE AND THE SNAKE BITTEN IN AUSTRALIA.[1]

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INTRODUCTION.

LAST year correspondence appeared in the columns of THE MEDICAL JOURNAL OF AUSTRALIA which indicated that the pioneer work of Martin and Tidswell on the Australian snakes was being forgotten and a discussion which I had with the Editor in Sydney on this matter, combined with previous experience regarding the great value of specific polyvalent antivenene in the treatment of cobra and Russell's viper bites in India led to my approaching Dr. Kellaway regarding the feasibility of reopening the study of the Australian colubrides in Melbourne.

Dr. Kellaway immediately took up the matter and obtained a grant from the Commonwealth Government without which the investigation could not have proceeded and more recently when our stocks of venom were adequate for the purpose, arrangements were made with Dr. Morgan, Director of the Commonwealth Serum Laboratories, to undertake the inoculation of horses with tiger and death adder venom. At the present time antivenenes for the two most deadly Australian snakes are in process of manufacture.

At the Walter and Eliza Hall Institute Dr. Kellaway commenced investigations on the titration and pharmacological action of various venoms in laboratory animals, while I undertook certain practical aspects of the ophidian problem which immediately required attention, such as the organization of the collection of venom, the investigation of the venom yields in different species of colubrides, their mechanism of bite and an experimental inquiry into the efficacy of ligature and other forms of local treatment in large sized animals.

More detailed information regarding the different phases of the investigation will be made available in a series of papers shortly to be published and only the general conclusions reached as a result of this work will be reviewed in the present paper, including the more important contributions made by past workers, especially in this country.

THE MORTALITY FROM SNAKE BITE.

In Table I will be found a record of the deaths occurring within the Commonwealth from venomous bites and stings which has been kindly supplied by Dr. J. H. L. Cumpston, Director-General of Public Health.

It will be noted that in the seventeen year period from 1910 to 1926, 244 people died from these causes, of whom 179 or 73·4% were males and 65 or 26·6% were females. The number of persons dying annually varied from six to twenty-nine, the average being approximately fourteen. The distribution of deaths in the different States is considered in Table II, Queensland having the greatest number and Western Australia the fewest.

TABLE I.

Deaths in the Commonwealth from Venomous Bites and Stings (1910 *to* 1926).

Year.	Deaths from Venomous Bites and Stings.		
	Males.	Females.	Total.
1910	8	4	12
1911	14	7	21
1912	10	6	16
1913	11	5	16
1914	16	5	21
1915	6	0	6
1916	11	2	13
1917	11	2	13
1918	4	3	7
1919	5	3	8
1920	18	5	23
1921	21	8	29
1922	19	4	23
1923	6	0	6
1924	1	5	6
1925	10	2	12
1926	8	4	12
TOTAL	179	65	244

TABLE II.

The Deaths in the Different States from Venomous Bites and Stings (1910 *to* 1926).

States.	Deaths from Venomous Bites and Stings.
Queensland	92
New South Wales	71
Victoria	54
Tasmania	10
South Australia	10
Western Australia	7
COMMONWEALTH	244

One death in the Federal Territory is included among the New South Wales figures and one death in the Northern Territory is included in the South Australian figures.

Taken in quinquennial periods, no evidence is obtained that the mortality is decreasing. Stings as well as venomous bites are included in these figures and if the snake bite mortality is taken as representing 80% of this total, it means that in the seventeen years under review 195 persons have died from this cause.

Though by no means formidable such a series of deaths does suggest the advisability of reviewing the present position regarding snake bite and the snake bitten in this country.

THE LETHALITY OF DIFFERENT SNAKES FOR MAN.

Of the one hundred and fifteen species of snakes found in Australia over seventy might be classed as venomous. Fortunately, however, only a few of these are capable of injecting a lethal dose of venom into man and those which do all belong to the proteroglyphous colubrides, that is colubrides with anterior grooved fangs.

The important Australian venomous snakes are the death adder (*Acanthophis antarticus*), the tiger snake (*Notechis scutatus*), the copper-head (*Denisonia superba*), the brown snake (*Diemenia textilis*) and the black snake (*Pseudechis porphyriacus*).

[1] Read at a meeting of the Victorian Branch of the British Medical Association on October 3, 1928.

In Tidswell's series[1] of 190 cases of snake bite ten were caused by death adders, thirty-two by brown snakes, thirty-three by tiger snakes, eighty-seven by black snakes and twenty-eight by non-specified species, the venomous character of the latter being evidenced by the presence of fang punctures. The mortality rate for the death adder was estimated as 50%, the tiger 45%, the brown snake 18·7% and the black snake 0%.

Ferguson[2] reviewed cases of snake bite occurring in New South Wales from 1906 to 1924 basing his analysis on police reports. Of 119 persons bitten seven died, but the incomplete nature of the returns is shown by the fact that over the same period forty-seven deaths from snake bite were recorded by the Government Statistician for this State. In other words forty fatal cases had occurred which did not appear in the police records and which in consequence were not available for analysis.

In Ferguson's series the species of snake responsible was the black snake in thirty-eight instances, the brown snake in thirty-eight, the tiger snake in twelve and the copper-head (*Denisonia superba*) in one. The other snakes listed were the green tree snake (*Dendrophis punctulatus,* two), bandy-bandy (*Furina occipitalis,* one), carpet (*Python spilotes* var. *variegatus,* one), and the Mallee snake (probably *Diemenia nuchalis*). Of the latter group the carpet is non-poisonous and the others only slightly venomous to man. In addition in twenty-three instances with three deaths the species was not ascertained.

Limiting observations to the known lethal snakes, the available evidence indicated that none out of thirty-eight brown snake bites, one out of thirty-eight black snake bites and three out of twelve tiger snake bites were fatal. The only person bitten by a copper-head recovered and there was no example of death adder bite in the series. Ferguson questioned whether the term black snake necessarily meant *Pseudechis porphyriacus* and information other than that contained in the police report made it appear likely that in one of the two fatal cases so reported a tiger snake was implicated. For this reason this case has been transferred to the latter category. In Victoria confusion also occurs and dark coloured copper-heads are liable to be described as black snakes by those who have not specially studied the question.

During the past year *questionnaires* from the Walter and Eliza Hall Institute have been sent to practitioners in different States regarding cases of snake bite which presented features of interest. Histories of twelve fatal cases and a number of non-fatal ones were obtained for clinical analysis, but as they were partly selected they cannot be utilized in assessing the mortality rate. The series included six deaths due to tiger snakes, four to death adders, one to the brown snake and one doubtful case of black snake bite from Queensland, the reptile in the latter instance not being procured for identification.

Tidswell's and Ferguson's figures constitute the only basis for estimating the mortality rate of different poisonous snakes in this country and in Table III the results of their conjoint surveys are presented.

TABLE III.

The Mortality Rate produced by the more common Venomous Australian Snakes. (Tidswell and Ferguson.)

Species of Snake.	Number of Persons Bitten.	Deaths.	
		Number.	Percentage.
Death adder ..	10	5	50
Tiger	45	18	40
Brown	70	6	8·6
Black	125	1	0·8
TOTALS ..	250	30	12

It will be seen that of the 250 cases of snake bite analysed, 125 were due to the black snake, seventy to the brown, forty-five to the tiger and only ten to the death adder and that the mortality rate in the same order was 50%, 40%, 8·6% and 0·8%. Thirty out of 250 instances of bites by these snakes proved fatal, giving a general mortality rate of 12%. The majority of these bites occurred in New South Wales, but as brown and black snakes are widely distributed throughout Australia, the incidence of their bite will be universally high. In Northern Australia, however, death adder bites are more frequent than these figures indicate, while further south in Victoria and Tasmania the copper-head would be more frequently implicated. In the combined series only one case (non-fatal) due to the latter snake was reported.

Martin[3] recorded death in a snake charmer within twenty-four hours of his being bitten by a large sized copper-head. The case is of special interest as the man had been bitten with impunity by *Notechis scutatus* on many previous occasions.

THE POTENTIAL DANGER OF DIFFERENT SPECIES ASCERTAINED EXPERIMENTALLY.

In the preceding section it was seen that the black snake which most frequently bites man in Australia, is rarely if ever the cause of death, whereas the mortality with the death adder and the tiger snake is probably higher than that recorded for any other terrestrial species.

In ascertaining the potential danger of any given species of snake to man the toxicity of its venom, the venom yield, the efficiency of its inoculating mechanism as well as its habitat and habits must all be taken into account.

The Toxicity of the Different Venoms.

Tidswell[1] injected progressively increasing doses of different venoms subcutaneously into rabbits and estimated that the certain killing dose for the tiger snake was 0·05 milligramme, for the death adder 0·2 milligramme, for the brown snake 0·2 milligramme and for the black snake 0·7 milligramme per kilogram of body weight.

For experimental work during the present investigation, I have used sheep which more nearly approach the body weight of man. The procedure in ascertaining the certainly lethal dose of black snake venom is shown in Table IV, while data for other venomous Australian snakes as well as for the cobra and Russell's viper are given in Table V. There it is shown that if the toxicity of cobra venom be taken as unity, then that of the tiger snake is twenty-five times, the death adder ten times, the copper-head two and a half times, the black snake one-third and the Russell's viper one-fifth as poisonous as this standard Indian venom for sheep.

Table IV.

Observations on the certainly Lethal Dose of Dried Black Snake Venom (Pseudechis porphyriacus) inoculated subcutaneously into Sheep.

Identification Number.	Weight in Kilograms.	Total Dosage in Milligrammes.	Dosage in Milligramme per Kilogram Body Weight.	Result.		
				Recovered.	Paralytic Time.	Death Time.
					Hours. Minutes.	Hours. Minutes.
73	42·5	2·13	0·05	Yes	—	—
75	43·1	4·3	0·10	Yes	—	—
72	44·1	6·6	0·15	Yes	—	—
64	44·5	8·9	0·20	Yes	—	—
73 (a)	42·5	17·0	0·40	No	25 10	106 10
75 (a)	43·1	25·9	0·60	No	3 54	27 43
93	38·2	22·9	0·60	No	13 4	13 59
79	40·7	24·4	0·60	Yes	—	—
89	41·1	24·6	0·60	No	17 15	18 15
72 (a)	44·1	35·3	0·80	No	4 25	24 3

The certain killing dose is approximately 0·8 milligramme per kilogram.
For the first four animals the dilution of venom was ten milligrammes per cubic centimetre, and for the last six it was twenty milligrammes per cubic centimetre.

Venom Yields.

Tidswell[1] recorded the average yields of dry venom for the tiger snake as 26·2 milligrammes (20 to 40 milligrammes), the death adder as 56·7 milligrammes (42·4 to 71·0 milligrammes), the black snake as 12·9 milligrammes (4·0 to 26·0 milligrammes) and the brown snake as 4·8 milligrammes (4·0 to 5·6 milligrammes). The death adder was revealed as the largest venom producer (two observations), while the brown snake proved much the smallest (thirteen observations). Martin[3] recorded maximal yields for the tiger snake as 73 milligrammes and for the black snake as 94 milligrammes of dried venom.

The results obtained during the present investigation are epitomized in Table V, the average total yield in the case of the cobra and Russell's viper being based on Acton and Knowles's published figures.[4] Their primary yields obtained by milking averaged 317·0 milligrammes in the cobra with variations of from 30·2 to 881·4 milligrammes and in the Russell's viper 108·0 milligrammes, the minimum individual yield being 60·0 milligrammes and the maximal 145·0 milligrammes of dried venom.

Owing to the habits of the Australian snakes in striking at inanimate objects or biting themselves when caught, the primary yields are often too low and for this reason in the case of the tiger snake, black snake and death adder the maximal yields observed in captivity for each snake were substituted for the primary yields. These averaged 47·2 milligrammes, 47·2 milligrammes and 84·7 milligrammes respectively. In the case of twenty-five copper-heads the average primary and maximal yields only differed by one milligramme, being 24·9 and 25·9 milligrammes respectively. The primary yields in a second series of fifty-six copper-heads of larger mean dimensions averaged 40·1 milligrammes, while the combined series of eighty-one snakes averaged 35·6 milligrammes at the first milking. This is the figure utilized for average total yield in Table V.

Two recently captured large brown snakes and six others of moderate size which had been in captivity for periods of from nine to twelve months, failed to give any venom on milking. The poison glands of these narrow-headed snakes are both relatively and absolutely much smaller than in the other species under consideration and this no doubt is related to the limited venom yields in captivity as well as to the lower mortality rate of its bites in man.

When both the quantity and the toxic quality of these venoms are taken into account, it is found that the average total yield of the tiger snake at one milking would suffice to kill one hundred and eighteen sheep, the death adder eighty-four, the cobra thirty-one, the copper-head eight, the Russell's viper two and the black snake one, while the maximal yield recorded for each species would kill 387, 235, 88, 47, two and two sheep respectively (Table V). The potential killing power of the tiger snake is thus 3·5 and of the death adder 2·7 times that of the Indian cobra (*Naia tripudians*), whereas the copper-head is only about one-quarter and the black snake one-twentieth as dangerous.

The Relative Efficiency of the Inoculation Mechanism in the Different Species.

Acton and Knowles[4] have emphasized the fact that in colubride snakes, owing to the anatomical attachments of the anterior temporal muscle to the mandible and its relationship to the venom gland, fixation of the lower jaw must occur if anything of the order of a full dose of venom is to be inoculated (see Figure I). The Australian colubrides are no exception to this rule, though when the

TABLE V.

A Comparison of the Relative Toxicity of Various Venoms for Sheep and of Their Killing Capacity estimated in terms of the Total Yields.

Species of Snake.	Average Total Yield in Milligrammes.	Certainly Lethal Dose in Milligrammes per Kilogram Body Weight.	Relative Toxicity for Sheep (Cobra venom = 1).	Certainly Lethal Dose for a Sheep of 40 Kilograms.	Number of Certainly Lethal Doses in Total Average Yield.	Number of Certainly Lethal Doses in Greatest Single Yield.
				Milligrammes.		
Naia tripudians (Cobra) ..	317·0	0·25	1	10	31·7	88·1
Viper russelli (Russell's viper)	108·0	1·25	0·2	50	2·2	2·9
Notechis scutatus (Tiger snake)..	47·2	0·01	25	0·4	118·0	387·5
Ancanthophis antarcticus (Death adder)	84·7	0·025	10	1·0	84·7	235·6
Denisonia superba (Copper-head)	35·6	0·1	2·5	4·0	8·9	47·4
Pseudechis porphyriacus (Black snake)	47·2	0·8	0·33	32·0	1·5	2·3

The figures of the total yields of the first two are given by Acton and Knowles, who record that for the cobra the mean yield was 317·0 milligrammes, the minimum 30·2 milligrammes, and the maximum 881·4 milligrammes, while for the Russell's viper they were 108·0, 145·0 and 60·0 milligrammes respectively.

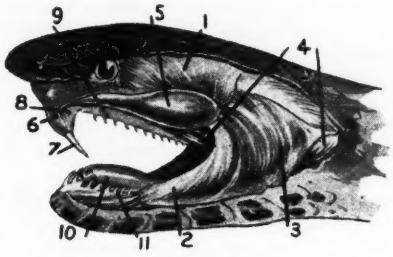


FIGURE I.
Death Adder.—Superficial dissection, showing poison gland.

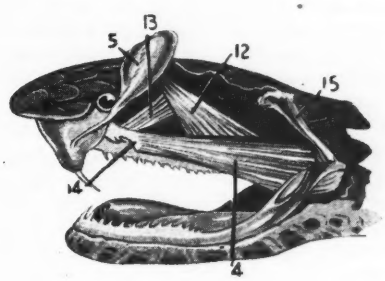


FIGURE II.
Death Adder.—Deep dissection, with gland everted.

FIGURE III.
Death Adder.—Jaws apart, showing the fangs, post maxillary and pterygopalatine teeth.

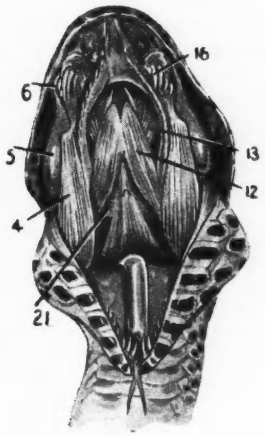


FIGURE IV.
Death Adder.—Dissection of the roof of the mouth, illustrating the essential muscles involved in the mechanism of bite.

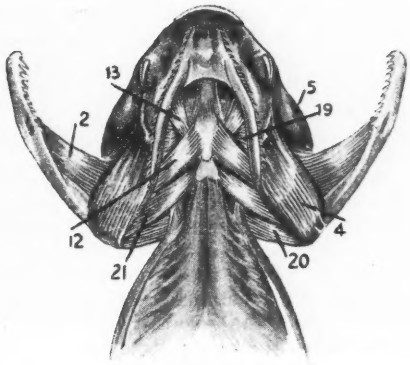


FIGURE V.
Tiger Snake.—Dissection of the roof of the mouth after separation of the mandibles.

DEATH ADDER AND TIGER SNAKE
(*Ancanthophis antarcticus*) (*Notechis scutatus*)

1. Anterior Temporal Muscle (Syn. Mandibular of Owen).
2. Anterior Temporal Muscle (Mandibular Portion).
3. Posterior Temporal Muscle.
4. External Pterygoid.
5. Poison Gland.
6. Poison Duct.
7. Grooved Fang.
8. Vagina Dentis.
9. Pterygo-Palatine Teeth.
10. Trachea.
11. Recurved Mandibular Teeth.
12. Spheno-Pterygoid Muscle.
13. Spheno- or Parieto-Palatine Muscle.
14. External Process of the Ectopterygoid into which the External Pterygoid Muscle is inserted.
15. The Quadrate Bone.
16. Reserve Fang.
17. Post-Maxillary Teeth.
18. Forked Tongue.
19. Parieto-Pterygoid Muscle.
20. Digastric Muscle.
21. Sub-Occipital Articular Muscle (Dugès).

glands are full, a quantity of their contents may be ejected under pressure during a snap bite when the snake fails to grip its prey entirely.

In an effective bite it is necessary for the bitten part to be firmly gripped with both jaws and failure to do so decreases considerably the chances of a lethal dose being injected. This, however, may take only a fraction of a second as when the limb is suddenly withdrawn, but at other times the snake hangs on with typical "bull dog" tenacity and in one of the fatal cases recently reported in our *questionnaire*, outside assistance was necessary before a tiger snake could be forcibly evulsed from the thumb of its victim.

The vipers possess to a remarkable degree the capacity to rotate the superior maxilla on its transverse axis of articulation with the prefrontal so producing elevation of its elongated canaliculated fangs and these two factors, length of fang and forward rotation, constitute the basis of an efficient biting mechanism.

The death adder is the only Australian colubride possessing lengthy fangs which average about six millimetres, whereas in the other species the fangs never appear to exceed five millimetres. In the copper-head and tiger the average length is about three millimetres, though in larger sized specimens they may equal five millimetres. The brown snake possesses the smallest fangs of the series.

These Australian snakes also possess to a variable degree the power of elevating and rotating forward the fangs, but the mechanism of its production differs essentially from that of *Viperidæ*.

In another paper this subject is dealt with in detail and a definite relationship is demonstrated between this power of forward rotation and the degree of maxillary shortening possessed by the different species. Thus, the maxilla is shortest in the death adder which possesses a maximal capacity to elevate and rotate the fangs, and longest in the brown snake whose powers in this direction are minimal.

From the standpoint of inoculation and biting efficiency the death adder easily ranks first. Then come the tiger, black snake and copper-head, while the brown snake must be placed last. Many of the characteristics of the latter snake link it with the non-poisonous colubrides, just as many of the features of the death adder show its close relationship to the vipers,

Commentary.

We are now in a position to consider the potential danger of these different species of snakes to man and it will be noted that the order follows closely the mortality rates based on Tidswell's and Ferguson's surveys.

Undoubtedly the death adder takes first place and on account of its efficient biting mechanism, its large poison yield and its potent neurotoxic venom it ranks as one of the most deadly of all known terrestrial snakes. Its average venom yield will suffice to kill eighty-four sheep. In biting efficiency it is definitely superior to the cobra on account of its greater capacity to rotate the fangs forward, while the potential killing power of its average venom yield is 2·7 times greater than that of *Naia tripudians* for sheep.

The tiger snake produces venom which is two and a half times more lethal for sheep than death adder venom and its superiority in smaller animals is even more evident. Its venom is certainly more potent per milligramme than that of any known snake and its average yield would kill one hundred and eighteen sheep. On account, however, of its lower venom yield, shorter fangs and their more limited forward rotation, it must be placed second in order of lethality.

The copper-head produces a powerful neurotoxic venom which is about two and a half times as poisonous as that of the cobra, though only one-tenth that of the tiger snake. The average yield would kill eight and the maximal individual yield forty-seven sheep. In venom production and inoculating efficiency it is inferior to *Notechis scutatus*. No statistical data regarding its mortality rate for man are available, but on experimental grounds it is listed third in order of lethality.

The brown snake has the least efficient biting mechanism, the smallest glands and the lowest venom yield of any of these snakes. The potency of its venom for rabbits was found by Tidswell[1] to approximate to that of the death adder, but no observations are available on its toxicity for larger sized animals. The small venom content of its glands and its short fangs account for the large number of recoveries following its bite, but when its venom is efficiently inoculated, death certainly may follow, as is shown by the numerous fatal cases recorded in the literature.

Finally, the black snake remains for consideration. Its poison yield approximates to that of the tiger snake and its inoculation mechanism is reasonably effective. Its venom, however, is much less potent than that of the other Australian snakes, being only one seventy-fifth as toxic as tiger snake venom for sheep. The average venom yield obtained by natural bite *plus* milking contained only one and a half certainly lethal doses for sheep and the maximal individual yield 2·3 lethal doses. Martin[3] has recorded a maximal yield of 94 milligrammes for this snake and this would contain approximately three lethal doses. These figures show how vastly inferior the potential lethality of the black snake is compared to the other colubrides and support the view that it rarely if ever kills an adult man, though probably it may be lethal to children of low body weight.

If snakes were capable of inoculating the whole contents of their venom glands into their victim, recovery would be very rare, but fortunately under the conditions of natural bite many factors operate which prevent this being accomplished, and in consequence even with the most deadly snakes sublethal doses are not infrequently injected.

The intervention of clothing, inaccurate striking, especially when a snake is moulting, inadequate

gripping of the bitten surfaces between the jaws with consequent non-fixation of the mandibles, insufficient elevation of the fangs and a poor venom supply at the time of striking are all factors causing ineffectual biting. Decreased yields may be caused by disease, moulting, recent feeding or biting and any given snake will show wide variations from time to time in the contents of its venom glands and in the quantity of poison it is capable of injecting into the tissues.

The whole clinical problem of snake bite revolves around the quantity of venom injected into the tissues and a more accurate appreciation of this fact would lead to less enthusiastic claims regarding the efficacy of various local and general therapeutic measures.

The Physiological Action of Snake Venoms.

Snake venoms contain a number of different constituents exerting divergent physiological effects. These include neurotoxins acting on the bulbar and spinal ganglion cells, hæmorrhagins destroying the endothelial cells lining the blood vessels, thrombase producing intravascular thrombosis, various cytolysins acting on red blood corpuscles, leucocytes and tissue cells, antifibrin or antocoagulin retarding the coagulation of the blood, antibactericidal substances, various ferments and kinases, the latter being of value in preparing ophidian food for pancreatic digestion. These toxic substances are differently distributed in various venoms, the vipers being characterized by the presence of hæmorrhagin, thrombase and cytolysins, whereas the colubrides typically contain neurotoxin, hæmolysin and anticoagulins.

The Australian colubride venoms of the black and tiger snake are of peculiar interest in this regard, for Martin and McGarvie Smith[5] demonstrated that in addition to potent neurotoxin they contained large quantities of thrombase, so constituting a sort of physiological linkage between the *Viperidæ* on the one hand and the *Colubridæ* on the other.

Our present investigations have confirmed Martin's results, but recent experiments have also shown that though neurotoxin and hæmorrhagin are universally represented, thrombase effects are not evident in sheep inoculated with death adder and copper-head venoms. Similar findings regarding copper-head venom are recorded by Kellaway who first noted the absence of intravascular thrombosis in small laboratory animals experimentally inoculated.

Chemical Considerations.

Mitchell and Reichert[6] pointed out the existence of two poisonous principles in snake venom, one acting on the central nervous system and the other being responsible for local lesions. The same observers showed that crotalus venom was non-dialyzable and lost all its hæmorrhagic properties when heated to 80° C. without destroying its neurotoxic constituents.

C. J. Martin[7] found that the venom of *Pseudechis porphyriacus* contained a diffusible thermostable albumose which was filterable through gelatine under fifty atmospheres of pressure and was responsible for respiratory failure, whereas a non-diffusible constituent which was coagulated at 85° C. produced hæmorrhages on injecting venom into animals. Chemically this last substance is now known to be of the nature of a globulin and thrombase also belongs to this category.

Hæmolysins on the other hand are very stable and have been found to resemble neurotoxins in most of their physical and chemical properties. Kyes[8] clearly differentiated the two constituents by extracting an aqueous solution of venom with a chloroform solution of lecithin, the resulting venom-lecithid which was separated by centrifugalization, being hæmolytic but not neurotoxic. In the original aqueous solution the whole neurotoxic properties of the venom were found to reside.

Acton and Knowles[9] point out that the viperine and the colubrine venoms may be differentiated by chemical means according to whether their toxicity lies mainly in the thermostable albumoses (neurotoxin and hæmolysin) or in the proteins coagulated by heat (hæmorrhagin and thrombase). They estimate that cobra venom contains 38% of coagulable protein, 50% albumoses and 12% enzymes, whereas Russell's viper venom shows 74%, 23% and 3% respectively.

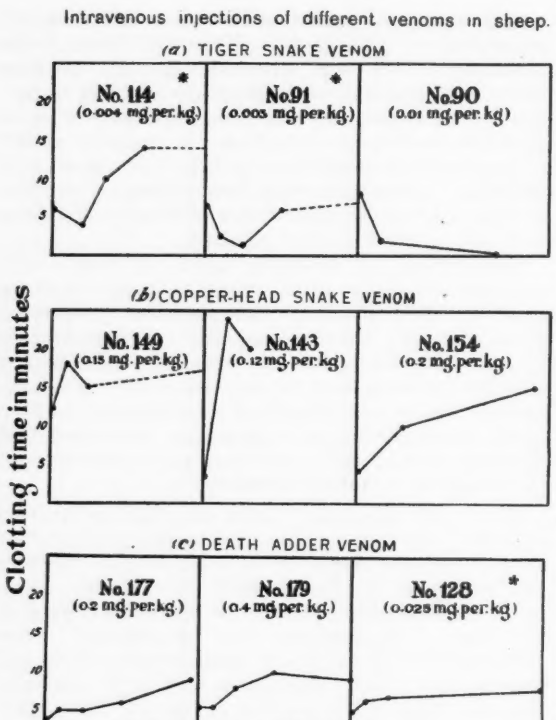
Neurotoxin.

Neurotoxin is the most potent constituent of colubrine venoms. It possesses a special affinity for the cells of the respiratory centre and the bulb, though it also involves nervous tissues elsewhere in the cord and brain.

Respiratory paralysis can be immediately produced in mammals by placing venom in the fourth ventricle and with black snake venom Martin[10] showed that after subcutaneous injection the circulation was well maintained up to the time that all respiratory movements ceased.

Acton and Knowles[9] consider the chemical linkage between neurotoxin and the nerve cell to be a loose one, capable of dissociation by specific antivenene. This is the reason why colubrine antisera can be given in fractional doses and is successfully administered as late as the end of the second third of the period from the time of biting to the date of the expected death.

Kilvington[11] studied the effects of tiger snake venom on the brain and spinal cord of rabbits and found the stigmata of toxic degeneration most evident in the cells surrounding the central canal of the cord. The cellular changes consisted of chromatolysis with degeneration and disappearance of the Nissl granules, loss of nuclear outline and finally disappearance of the nucleus itself. Inflammatory and vascular changes were absent. Lamb and Hunter[12] reported detailed investigations on the action of venom of different poisonous snakes on the whole central nervous system.



GRAPH I.

which clotting was produced. Intravascular thrombosis occurred when a small quantity was given intravenously or a relatively large amount of venom was injected subcutaneously into small animals. Thrombosis was most readily produced in the portal vein and its tributaries and sometimes this was found to be associated with decreased coagulability in the systemic vessels. Further when the venom was sufficiently concentrated, death from circulatory stasis invariably ensued, but when the concentration fell short of that necessary to raise the coagulability of the blood to such an extent as to occasion thrombosis, the blood shortly afterwards lost its capacity to clot. In this condition a sort of negative phase had been established and further injection of venom failed to produce clotting.

Martin pointed out the analogy existing between his results and those obtained by Wooldridge after injecting nucleo-proteins intravenously and suggested that the lytic action of venom on tissue cells might determine the presence of such substances in the blood stream.

Lamb[14] found that citrated plasma or blood was coagulated by Russell's viper venom *in vitro*. Martin[15] reinvestigated the question of venom coagulation modifying his original views concerning the mechanism of thrombosis. He found that coagulation of citrated or oxalated plasma was independent of calcium ions and that the fibrin ferment contained in the venom did not appear to be used up in the process.

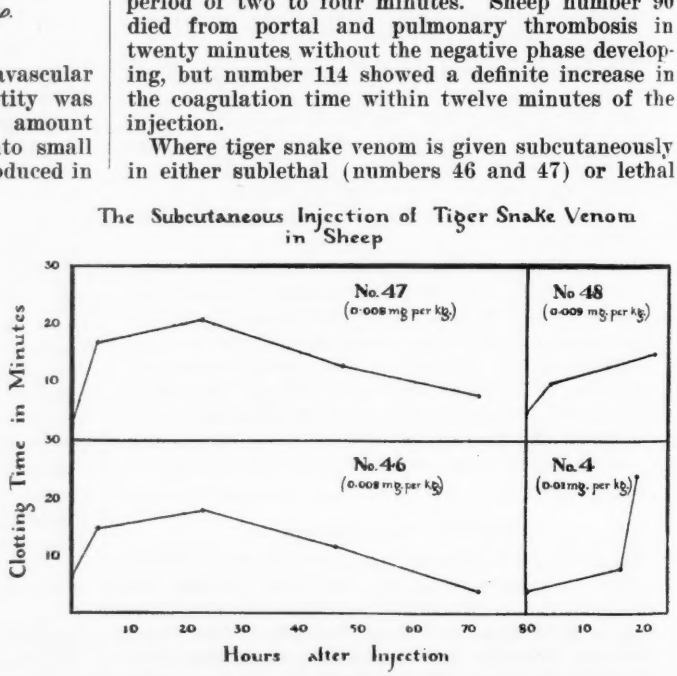
Houssay and Negrette[16] investigated the coagulant action of venoms both *in vitro* and *in vivo* and came to the conclusion that the positive phase of increased clotting was due to the precipitation of fibrinogen by the venom itself and that the negative phase was a consequence of this precipitation and not due to the formation of antithrombin as had been previously suggested.

Effects of Thrombase Containing Venoms in Sheep.

The two thrombase containing venoms investigated in ovines were those of the Russell's viper and the tiger snake and the findings of other observers regarding the rapid establishment of increased coagulability after intravenous injection followed by a period of decreased coagulability were fully confirmed.

In Graph I the coagulation times in sheep numbers 114, 91 and 90 are plotted over a period of twenty minutes after 0·004, 0·003 and 0·01 milligramme of dried tiger snake venom per kilogram body weight had been injected intravenously. In all three animals a very definite decrease in the coagulation time of the blood occurred within a period of two to four minutes. Sheep number 90 died from portal and pulmonary thrombosis in twenty minutes without the negative phase developing, but number 114 showed a definite increase in the coagulation time within twelve minutes of the injection.

Where tiger snake venom is given subcutaneously in either sublethal (numbers 46 and 47) or lethal



GRAPH II.
The negative phase of decreased coagulability of the blood produced by the subcutaneous injection of tiger snake venom in sheep.

Neurotoxic Effects in Sheep.

Death adder, copper-head, tiger and black snake venoms all prove powerfully neurotoxic in ovines and muscular twitchings, paresis, ataxia and later paralysis of the fore and hind limbs ensue when a lethal dose has been injected. The trunk and neck muscles subsequently become involved to a variable degree.

Progressive bulbar paralysis is a characteristic finding in sheep, the pendulous lower lip, dribbling saliva, the protruding flaccid tongue combined with an inability to swallow constituting a remarkable and constant picture in the terminal stages. Owing to palatal paralysis the regurgitated rumen contents may be emitted from the nostrils, while loss of the sensory reflex from the larynx (ninth nerve) also permits its aspiration through the glottis into the bronchial tree and bronchioles which may be found at autopsy to be tightly packed with food *débris*.

Paralysis of the pharyngeal muscles also prevents saliva being swallowed. In consequence it collects in quantity in the mouth, naso-pharynx and nasal cavities, welling out through the nostrils or dribbling through the paresed lips according to the position of the head. Increased salivary secretion probably also occurs.

Respiratory failure is the common mode of death with all colubrine venoms and sheep die regularly from this cause. Lack of thoracic movements in the latter stages is characteristic, though urgent dyspnœa may be observed at earlier periods. Involvement of the spinal accessory nucleus in the medulla with consequent paralysis of the sterno-mastoid and trapezius renders fixation of the shoulder impossible and in consequence contraction of the pectorals and the serratus merely draws the shoulder on to the thorax, pulmonary ventilation being further impeded.

Death adder and tiger snake venoms produce much the same neurological features, but in copper-head poisoning the early onset of paralysis of the muscles of the neck leads to a pathognomonic picture of pathetic helplessness, the head rolling into a position determined by gravity. Respiratory distress in the earlier stages is less evident probably because of the profound and widespread nature of the paralysis involving the trunk muscles. These animals lie absolutely motionless for hours, dying quietly, despite well established bulbar paralysis and respiratory failure. One sheep for five days after the onset of paralysis was unable to stand and in this animal for several days subsequently muscular tremor and ataxia were noted.

Hæmorrhagin.

Local hæmorrhage and hæmorrhages scattered through the viscera have long been recognized in necropsies on snake bite cases. Mitchell and Reichert[6] investigated the effects of crotalus venom directly applied to the mesentery of warm blooded animals and showed that these extravasations were due to a direct solution of continuity of the capillary wall. Flexner and Noguchi[13] named this substance hæmorrhagin and showed that it was the chief death dealing constituent in many viperine venoms. They found that 90% of the toxicity of crotalus venom was attributable to hæmorrhagin, which differed fundamentally from neurotoxin in the intensity of its local effects. On direct application to the brain it proved twenty times more poisonous than when injected subcutaneously. Pure neurotoxin was equally lethal by either route.

Martin[10] studied the effects of hæmorrhagin in dogs inoculated with black snake venom. Hæmorrhages in the lung were constantly present, sometimes considerable areas being solidified from this cause, while radial hæmorrhages in the cortex of the kidney were not uncommon.

Hæmorrhagin Effects in Sheep.

In sheep inoculated with lethal doses of tiger snake, death adder, copper-head or black snake venom multiple hæmorrhages are almost invariably demonstrable in the lungs, the thymus and the small or large bowel. Other common sites are the endocardium, pericardium, suprarenals, thyreoid, pancreas and subcapsular zone of the liver. Occasional hæmorrhages in the bladder and kidneys are met with, but more frequently these organs are only congested. The largest hæmorrhagic lesions are found in the lungs and in the subperitoneal tissue at the base of the mesentery.

The selective affinity of hæmorrhagin for endothelium is well shown after the intravenous injection of a fatal quantity of tiger snake or Russell's viper venom in sheep in which hæmorrhagic infiltration of the endocardial lining of the ventricles is very striking.

Discoloration of the skin, a variable degree of swelling and petechiæ surrounding the fang punctures may occur. Subcutaneous hæmorrhage in the vicinity of the bite may also be met with especially in the death adder, black snake and tiger snake bites. The extravasation, however, is rarely extensive and never approaches in intensity the tremendous red current jelly infiltration involving the subcutaneous tissues after inoculation with viperine venoms (*Vipera russelli*). Local œdema is generally present and not infrequently it is gelatinous in type especially in copper-head poisoning. Unlike hæmorrhage it results from the action of the thermostable albumose fraction of the venom. These changes take some time to develope and local features including fang punctures are by no means always demonstrable.

Thrombase.

In the time at my disposal it is impossible to do justice to the complex problems involved in the coagulating and anticoagulating properties of snake venoms.

C. J. Martin[10] who two years previously had demonstrated the thrombotic action exerted by the venoms of *Pseudechis porphyriacus* and *Notechis scutatus*, investigated in 1895 the mechanism by

(numbers 48 and 4) doses decreased coagulability may become very evident (Graph II). The first two animals received 0·008 milligramme per kilogram and decreased coagulability persisted for at least fifty hours, while the last two showed a well established negative phase up to the time of death. Sheep number 48 became paralysed in twenty-one hours and forty minutes and died in twenty-three hours and ten minutes, while sheep number 4 developed paralytic features in ten hours and twenty-seven minutes and succumbed in nineteen hours fifty seven minutes.

Post Mortem Findings.

The intravenous certainly lethal dose of tiger snake venom for sheep was estimated at 0·005 milligramme per kilogram, but some animals succumb to quantities as small as 0·002 milligramme per kilogram. Animals which receive only one lethal dose, generally die in one to four hours from the effects of thrombosis involving the portal vein and its tributaries, and a very typical picture characterized by intense congestion of the mesenteric veins, multiple hæmorrhages in the entero-colon and stomach and marked splenomegaly results. Within four hours the spleen may increase in weight to over 400 grammes, its normal weight in sheep being 80-90 grammes and in these cases the splenic vein is always found clotted as well. Animals receiving larger quantities, *id est* 0·0075 to 0·02 milligramme per kilogram, generally die in about twenty minutes or less and at autopsy intracardiac thrombi involving the cavities of the right auricle and ventricle are almost invariably demonstrable. A tangled skein of fibrin whipped out by cardiac contractions is found around the tricuspid valves, while *ante mortem* clotting in the branches of the pulmonary artery is also characteristic. Only rarely does intravascular thrombosis occur in the systemic vessels and then only when very large doses of venom have been administered by the intravenous route.

In snake bite thrombase plays an important rôle in killing small prey such as birds and mice rapidly, but in large sized animals including man its action in this direction is insignificant. Neurotoxin is the killing constituent in all Australian colubride venoms as far as man is concerned.

Anticoagulins.

Halford[17] was the first observer in Australia to draw attention to the fluidity of the blood in man and animals following fatal bites by the tiger snake, while Martin[10] investigated the problem in detail as already outlined.

The negative phase of decreased coagulability manifest by thrombase-containing venoms is, however, an entirely different phenomenon to the one under discussion, for only the positive phase of increased coagulability can be demonstrated *in vitro*.

In the presence of certain other venoms, however, including that of the cobra, oxalated and citrated serum fails to clot on the addition of calcium ions, while injection into animals also retards the coagulation time or actually prevents clotting altogether.

Lamb and Hunter[12] showed in rabbits that intravenous injections of cobra venom even in large quantities entirely failed to produce intravascular thrombosis, but caused some deficiency in the coagulability of the blood, the extent of the latter being largely determined by quantitative considerations.

Calmette[18] held that the anticoagulant action of colubride venom was due to the actual destruction of fibrin ferment and later to proteolysis of fibrin, whereas Houssay and Negrette[16] attribute it to destruction of thrombokinase.

During the present investigation similar findings to those of Lamb and Hunter have been recorded in sheep injected intravenously with copper-head venom. When death resulted it was always due to neurotoxic effects and never to *ante mortem* portal or pulmonary thrombosis.

In Graph I the coagulation times of the blood of three sheep, Numbers 149, 143 and 154, are plotted over a period of twenty minutes following intravenous injection of 0·15, 0·12 and 0·2 milligramme of copper-head venom per kilogram. They died in three hours fifty-four minutes, forty hours forty-nine minutes and two hours twenty minutes respectively.

Normally in ovines the coagulation time does not exceed thirteen minutes and generally it varies from four to eight minutes, but in each of these animals an increase of from fifteen to twenty-four minutes occurred and the clot was of loose gelatinous type. No evidence of a positive phase of increased coagulability was observed and taken in conjunction with the absence of *ante mortem* thrombosis these findings indicate that thrombase is absent and that some anticoagulin constituent is present in copper-head venom.

In animals dying from intravenous injections of death adder venom evidence of *ante mortem* thrombosis is similarly absent. Graphs of the coagulation times in sheep Numbers 177, 179 and 128 which received 0·2, 0·4 and 0·025 milligramme per kilogram intravenously fail to show any positive phase of increased coagulability (Graph I), a result which again indicates an absence of thrombase. Over the twenty minute period a decreased coagulability of the blood was demonstrable in each case, but it never exceeded the upper limit of normality. Sheep Numbers 177 and 179 died in forty-one and thirty-five minutes respectively, while No. 128 recovered. These results combined with those obtained at autopsy show definitely that thrombase is absent from this venom, but the evidence that anticoagulin is present is less convincing.

Hæmolysins.

A hæmolysin producing lysis of the red blood corpuscles in the presence of complement or lecithin derived from the serum or the corpuscles themselves is one of the common constituents of snake venom.

Martin[10] studied the effects of black snake venom on the whole blood of different animals. The cor-

puscles of some species were found to hæmolyse readily, others not at all. Dogs were very susceptible and severe intravascular hæmolysis resulting in the laking of 50% of the circulating corpuscles was actually observed within a period of forty-eight hours when subminimal lethal doses were injected. Hæmoglobinuria frequently occurred and in animals dying with suppression of urine within two to three days of injection, microscopical examination showed the renal tubules to be completely blocked with hæmoglobin crystals. These were also demonstrable in the bile.

Flexner and Noguchi[13] showed that in some instances washed corpuscles were not hæmolysed by venom, but that this property could be restored by the addition of complement. This was destroyed at 56° C. and by tryptic digestion, but was not inhibited by cholesterin. Kyes[19] found that serum regained in enhanced degree this activating action on heating from 65° to 100° C. and chemical analysis showed it to be dependent on the liberation of lecithin. Kyes found two kinds of corpuscles, one type hæmolysing with pure venom and containing its own thermolabile activator, so called endocomplement, the other needing either extraneous complement or lecithin (cobra-lecithid) to enhance the affinity of the venom for the corpuscles.

Welsh and Chapman[20] studied the hæmolytic action of the venoms of different Australian snakes, including the black, brown, tiger and death adder on the corpuscles of several species of animals. They found that venoms varied greatly in their hæmolytic effects and that though complement did not activate washed corpuscles of unsusceptible species, it increased hæmolysis in susceptible corpuscles. Serum heated to 100° C. also accelerated hæmolysis in susceptible corpuscles and in some instances induced hæmolysis in unsusceptible, a result depending on the liberation of lecithin by heat.

With all these venoms occasional evidence of the action of hæmolysin was observed at autopsy on sheep, such as a pinkish-red staining of the aorta and endocardium and a diffusion zone of blood pigment around the terminal loops of the mesenteric vessels in the small intestine. Distension of the gall bladder with bile was also a very constant finding and it is not improbable that this is related to an excessive bilirubin excretion resulting from intravascular hæmolysis. As in man, however, hæmolysis was seldom a prominent feature. This, as Martin[3] has shown, is attributable to the fact that the dose of venom necessary to destroy life by respiratory paralysis is so very much less than the quantity required to produce extensive laking of blood cells.

Commentary.

In the foregoing section I have briefly reviewed the constituents found in the more common Australian colubride venoms as observed in inoculated sheep. These results are summarized in Table VI, but it should be noted that as the observations are based on a single species of animal, subsequent investigations may lead to their modification. Owing to the variable reaction of different species of animals to the different constituents of a venom as well as to the venom as a whole, positive results are of more value than negative, when only one species is under investigation.

TABLE VI.

The Constituents of Four Common Australian Colubrine Venoms observed in Sheep.

Species of Snake.	Neurotoxin.	Hæmorrhagin.	Hæmolysin.	Thrombase.	Anticoagulin.
Black snake	+	+	+	+	0
Tiger snake	+	+	+	+	0
Death adder	+	+	+	0	?
Copperhead	+	+	+	0	+

CLINICAL FEATURES OF SNAKE BITE.

Tidswell[1] has given us much the most accurate analysis of important data bearing on the clinical aspects of snake bite and his published figures are widely utilized in the following section.

Both Tidswell's[1] and Ferguson's[2] series showed that snake bite occurs in the summer months from October to March, though isolated cases have been recorded during the winter. This finding is related to habits of hibernation and the inevitable lethargy of cold blooded animals at low temperatures.

Age and Sex.

The age and sex in fifty fatal snake bites compiled from Tidswell's and Ferguson's series and partly from the present series showed that thirty-nine or 78% were met with in males and thirteen or 26% in females. Eighteen or 36% occurred in children under ten years, eight or 16% between the ages of eleven and twenty and the remaining twenty-six or 52% in adults over twenty-one years. The high proportion of fatal bites in the first decade of life should be noted.

Anatomical Location of the Bites.

A survey of the areas of the body bitten is of importance as on it depends the feasibility of ligature, as well as the institution of measures directed to lessen the incidence of bites. For this purpose the distribution of bites as determined by fang punctures in 281 cases occurring in Ferguson's, Tidswell's and the present series are detailed.

One hundred and fifty-nine out of the 281 persons or 56.6% were bitten on the lower extremity; of these bites twenty-one occurred on the toes, fifty on the feet, eighty-three on the legs and five on the thigh or knee. In 118 persons or 42% the upper extremity was bitten, the fingers being involved in seventy-two instances, the hands in seventeen, the forearm in twenty-one and the upper arm in eight. Of the remaining bites, two were located on the body, one on the neck and one on the face.

It will thus be seen that it is almost invariably the extremities which are bitten and that ligature can be employed as a first aid line of treatment in the vast majority of cases.

The Local Lesion.

The local lesions caused by the Australian venomous snakes are slight as compared to the *Viperidæ* and generally the systemic disturbances are altogether disproportionate to the local pain and swelling observed. The latter features increase in intensity after the application of ligature.

The two classical fang punctures separated by a distance of from one to two centimetres are present as a rule, but one, three or even four fang marks may sometimes result from the one bite. Skin scratches are frequent and marking due to the anterior mandibular or palatine teeth may occasionally occur. The fang punctures may be outlined by small areas of hæmorrhage, but the actual swelling of the tissues is due to an œdema rather than to local extravasation of blood, although this is sometimes present. Oozing of blood through the punctures is rarely observed, but discoloration of the skin is often present. Sometimes the fang punctures are not demonstrable, especially immediately following the bite.

Local suppuration is uncommon and gangrene so characteristic of viperine bites is of extreme rarity. Chronic ulcers are seen, but these are generally due to potassium permanganate treatment rather than to snake venom.

Systemic Manifestations.

The time of onset of symptoms will naturally be determined by the dose of venom injected and the rate of its absorption. The first symptoms may appear from a quarter to two hours after the bite and are characterized by nausea, faintness, vomiting, headache, lethargy and drowsiness. The various symptoms may be classified as follows.

Neurotoxic Symptoms.

Muscular weakness and paresis of the limbs, ataxia and a drunken gait are amongst the earliest neurological symptoms indicative of cord involvement. Definite paralysis of the limbs may supervene and in sheep its onset in fatal cases varied from one to forty-four and two-third hours with different venoms according to the amount injected. Frequency of micturition and a desire to defæcate may also be noted. Then follows difficulty in swallowing and saliva which may be either thick and tenacious and difficult to expel or of more fluid consistency, tends to collect in the nasopharynx. Speaking also becomes blurred and indistinct and sometimes a nasal quality is present owing to palatal paralysis. As this progresses mucus or vomitus may be regurgitated through the nasal passages.

As in ovines bulbar paralysis underlies many of the clinical features of snake bite and often it is ascending in type. Ocular involvement is not uncommon and ptosis, diplopia and dilated pupils may be observed. In the latest stages the pupils become widely dilated and insensitive to light. Coma and convulsions may terminate the picture, the latter being of asphyxial origin in most cases.

Respiratory involvement is invariable and in the earlier stages dyspnœa and air hunger may be severe. Later, breathing becomes shallower, little or no thoracic movement occurs, cyanosis increases and death results from respiratory failure, while the circulatory mechanism is still functioning. Artificial respiration under these circumstances may maintain life for several hours.

Cardio-Vascular Symptoms.

Cardio-vascular symptoms due to vasomotor and cardiac depression are characterized by subnormal temperature, rapid, feeble, thread-like pulse, cold extremities, sweating, blanched skin, nausea, vomiting and low blood pressure. Fear as emphasized by Creed[21] may be another factor contributing to this condition and a similar picture may follow a bite given by a non-poisonous snake. In many cases the blood pressure is well sustained and it is rare to observe in Australian snake bite the extreme grade of prostration characteristic of viperine bites. The venom of *Vipera russelli* exerts a direct toxic action on the vasomotor centre in the medulla and in sheep inoculated with it acute syncopal attacks associated with vasomotor failure are commonly observed.

Hæmorrhagic Extravasations and Clotting Time.

Small hæmorrhages into the mucous membranes are frequently observed at autopsy and clinically bleeding of the gums, epistaxis, hæmatemesis and hæmoptysis may occur.

Two instances of hæmaturia, one of hæmatemesis and one in which bright blood was passed *per rectum* were noted in the present series of twelve fatal cases. As a general rule these features are not prominent and their appearance is of grave prognostic significance.

Where local treatment has effaced the fang marks, it is often difficult to determine whether or not the patient is suffering from the bite of a venomous snake and under these circumstances a specimen of urine should always be examined for red cells and pigment. The kidney may leak blood in small quantities before it appears in the lumina of the hollow viscera and in a doubtful case of *Echis carinata* bite I was once able to make an early diagnosis by this means.

The coagulation time of the blood has been rarely estimated by clinical means, but it is worth ascertaining both on account of its diagnostic and prognostic significance. Decreased but never increased coagulation occurs at autopsy, the positive phase with the thrombase containing venoms not having been observed in man.

Clinical Features with Different Species. (Case Histories.)

A differential diagnosis between various species of colubride bites cannot be made clinically and

ILLUSTRATIONS TO THE SYMPOSIUM ON SNAKE BITE.

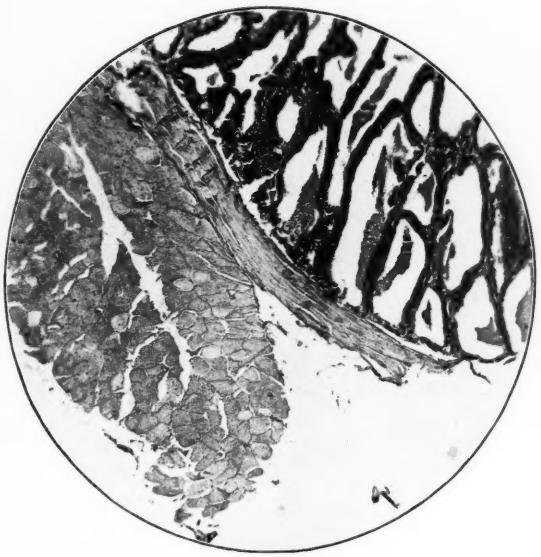


FIGURE VI.
1. Venom Gland (Tiger Snake).

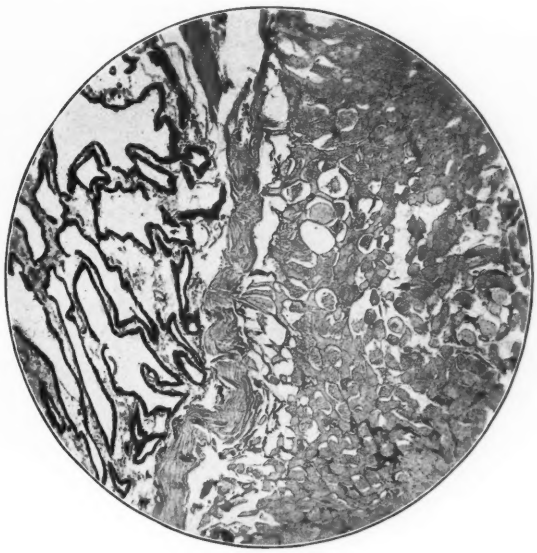


FIGURE VII.
2. Venom Gland (Death Adder).

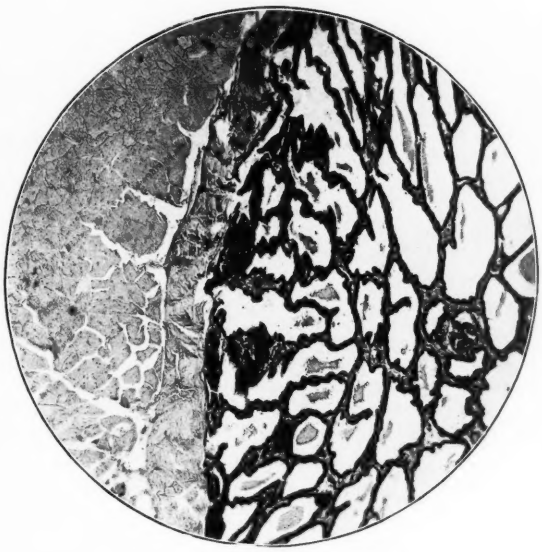


FIGURE VIII.
3. Venom Gland (Brown Snake).

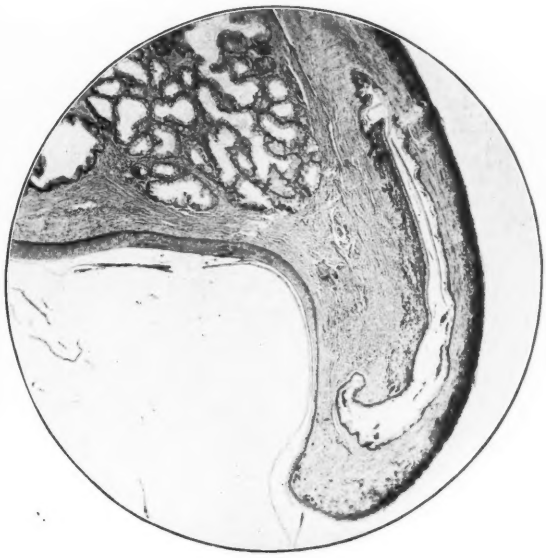


FIGURE IX.
4. Transverse Section of Venom Duct (Death Adder).

ILLUSTRATIONS TO THE SYMPOSIUM ON SNAKE BITE.

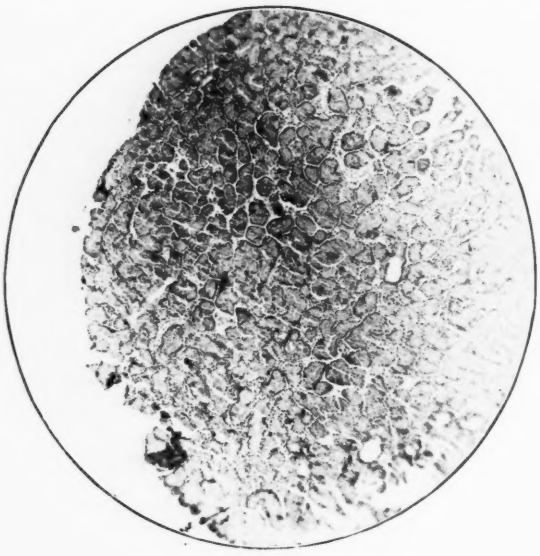


FIGURE X.
5. Maxillary Gland (Carpet Snake).

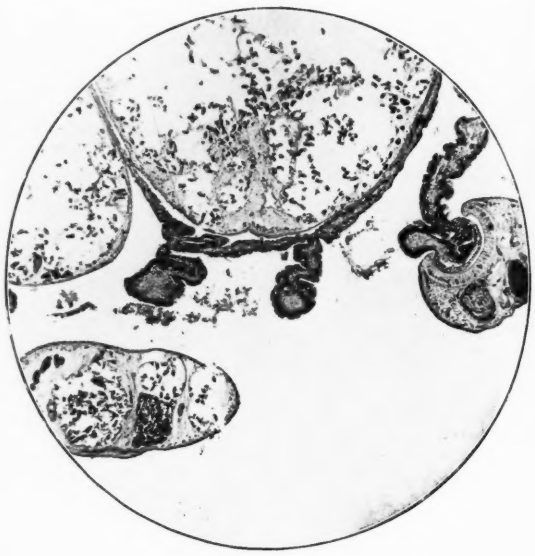


FIGURE XI.
6. Fluke in Lung of a Tiger Snake.

FIGURE XXIV.

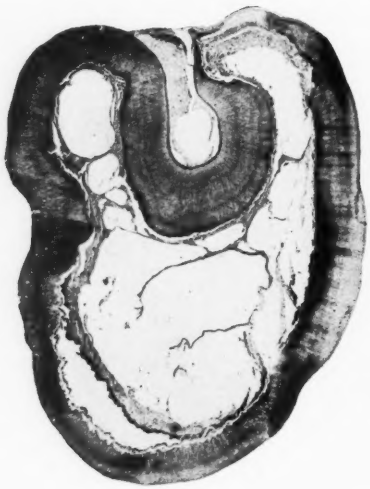


FIGURE XXV.

FIGURE XXVI.

FIGURE XXVII.

FIGURE XXVIII.

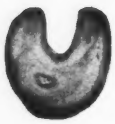


FIGURE XXIX.

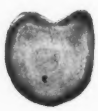


FIGURE XXX.

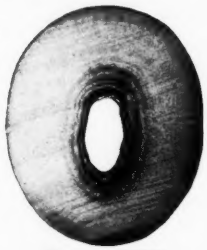


FIGURE XXXI.

Figures XXIV to XXX are microphotographs of serial sections through the fang of a death adder from base to apex, showing the anteriorly situated poison groove. Figure XXXI is through the tooth of a non-poisonous carpet snake, possessing a central pulp canal and dense dentine, but no grooving.

A Life-sized Reproduction of a Colour Photograph of the Death Adder (*Ancanthophis antarcticus*).

ILLUSTRATIONS TO THE SYMPOSIUM ON SNAKE BITE.

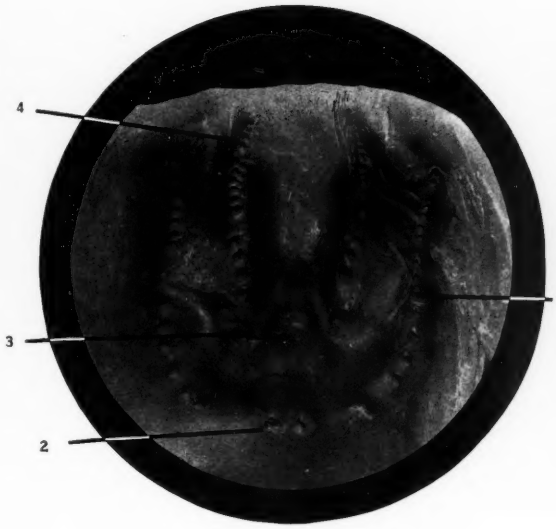


FIGURE XXXIV.
Carpet Snake.

FIGURE XXXV.
Diamond Snake.

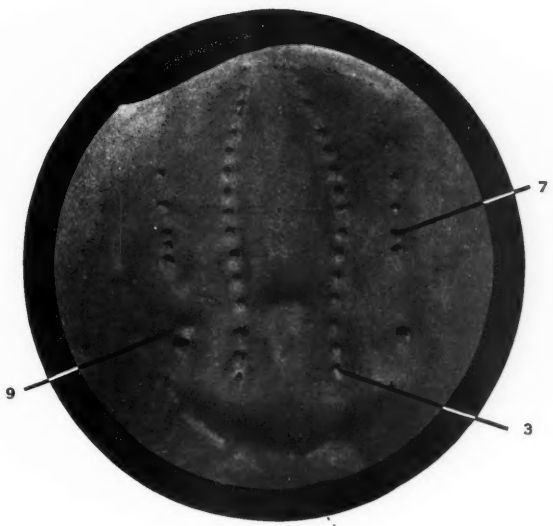


FIGURE XXXVI.
Brown Snake.

DENTAL IMPRESSIONS OF THE BITE OF DIFFERENT AUSTRALIAN SNAKES TAKEN IN KERR'S IMPRESSION COMPOUND.

Note the numerous maxillary teeth marks in the non-poisonous snakes and the prominent fang marks and scanty post-maxillary teeth in venomous colubrides.

1. Maxillary Teeth.
2. Premaxillary Teeth.
3. Palatine Teeth.
4. Pterygoid Teeth.
7. Post-maxillary Teeth.
9. Reserve Fang.

ILLUSTRATIONS TO THE SYMPOSIUM ON SNAKE BITE.

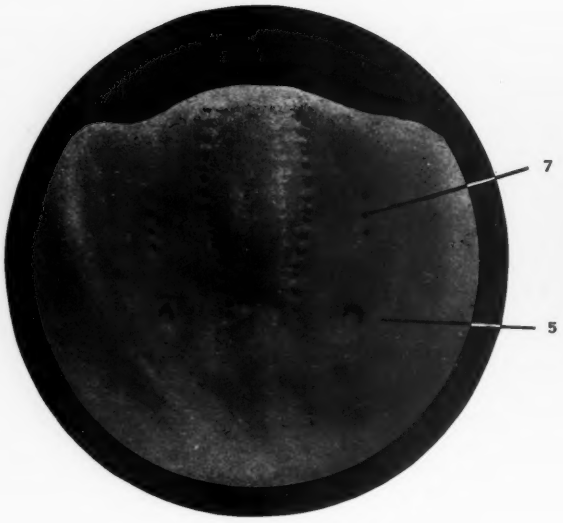


FIGURE XXXVII.
Copper-head Snake.

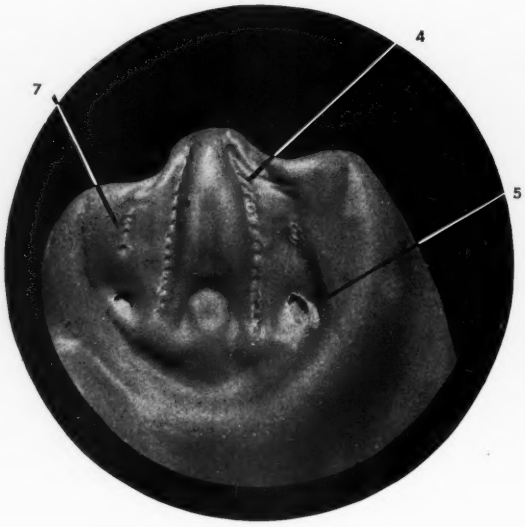


FIGURE XXXVIII.
Black Snake.

FIGURE XXXIX.
Death Adder (Lower Jaw).

DENTAL IMPRESSIONS OF THE BITE OF DIFFERENT AUSTRALIAN SNAKES TAKEN IN KERR'S IMPRESSION COMPOUND.

Note the numerous maxillary teeth marks in the non-poisonous snakes and the prominent fang marks and scanty post-maxillary teeth in venomous colubrides.

4. Pterygoid Teeth.
5. Fang Puncture.
7. Post-maxillary Teeth.
8. Mandibular Teeth.

ILLUSTRATIONS TO THE SYMPOSIUM ON SNAKE BITE.

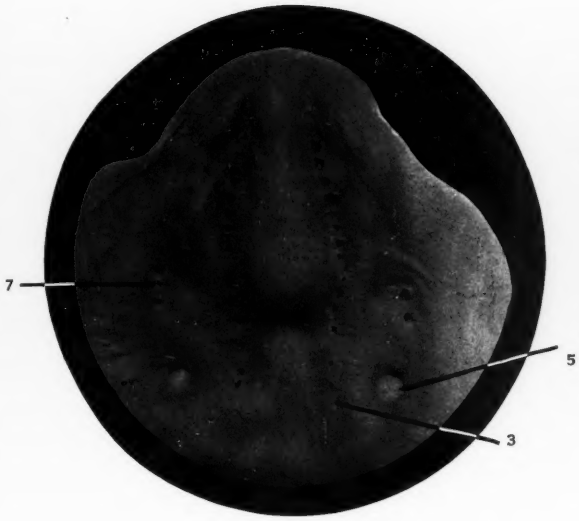


FIGURE XL.
Tiger Snake.

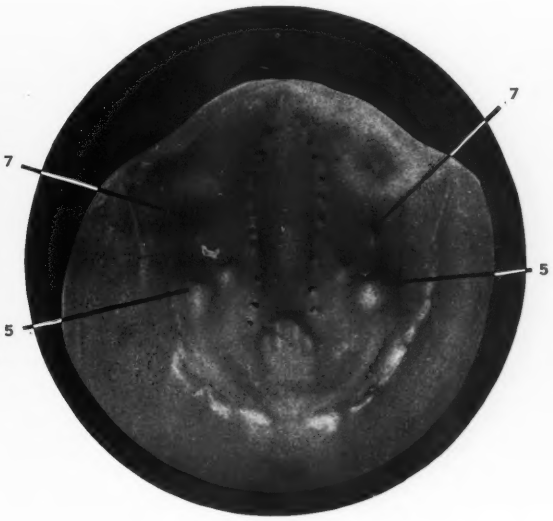


FIGURE XLI.
Death Adder (Two Fangs).

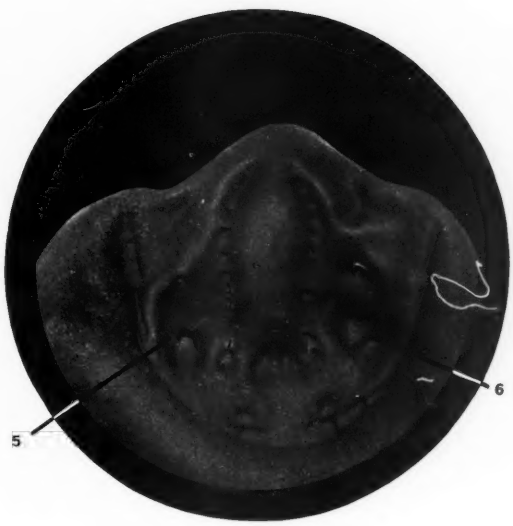


FIGURE XLII.
Death Adder (One Fang).

DENTAL IMPRESSIONS OF THE BITE OF DIFFERENT AUSTRALIAN SNAKES TAKEN IN KERR'S IMPRESSION COMPOUND.

Note the numerous maxillary teeth marks in the non-poisonous snakes and the prominent fang marks and scanty post-maxillary teeth in venomous colubrides.

3. Palatine Teeth. 5. Fang Puncture. 6. Absent Fang. 7. Post-maxillary Teeth.

different patients bitten by the same species of snake may exhibit considerable variations in the clinical picture. The following case histories illustrate the clinical features in different species.

Tiger Snake Bite.

Case I.—This patient had a drunken gait and was brought to the Melbourne Hospital in October last by the police. He was unable to talk, but wrote down that he was twice bitten while handling a tiger snake some six and a half hours previously, first on the ankle, then on the left hand. At the time he was selling a patent cure for snake bite. The punctures on the hand had been scarified by a razor and a ligature applied to the forearm. On the right ankle four punctures about twelve millimetres (half an inch) apart were noted. The skin in the vicinity showed purplish discoloration. The temperature was 35° C. (95° F.), the pulse rate 120 and the respirations were sixteen per minute, the systolic blood pressure was 115 millimetres of mercury and the diastolic 80. The patient presented a drunken appearance; he could make a noise, but not speak. The tongue was swollen and œdematous. The knee jerks were hyperactive, the superficial abdominal reflexes were present and the plantars gave a flexor response. The pupils were dilated and later failed to react to light. Paralysis of the limbs never developed. The patient became irritable, fibrillary twitchings of the tongue were present and some degree of muscular rigidity occurred. About eight hours after admission, that is fourteen and a half hours after being bitten, respiratory distress and cyanosis were marked and some two hours later death from respiratory failure ensued.

Autopsy by Dr. Mollison showed that *rigor mortis* and *post mortem* staining were marked. The large veins and also the heart which was relaxed, contained fluid blood. Congestion of the liver, kidneys, brain and lungs was found and the latter organ contained multiple small hæmorrhages. Locally ecchymoses were found surrounding the four bites on the ankle. The appearances were those of an asphyxial mode of death.

Death Adder Bite.

Case II.—This patient, a male, aged two years and eleven months, was admitted to the Chinchilla Hospital in March, 1928, under the care of Dr. K. J. Hill.

There was a history of the patient having been bitten on the inner side of the right foot below the internal malleolus. Two puncture marks had been noted by the parents who incised the wounds, rubbed in crystals of potassium permanganate and applied a ligature to the leg. The temperature was 36·6° C. (98° F.), the pulse rate 156 and the respiration 26 per minute. Later both the respiratory and pulse rate slowed. Pallor, sweating, vomiting and low blood pressure were present. The palate became paralysed and brownish fluid was vomited through the nose, the discoloration probably being due to altered hæmoglobin (hæmatin). Slight convulsive spasms developed and the patient died in a comatose condition from cardiac and respiratory failure three and a half hours after being bitten.

Case III.—Miss C., aged twenty-five years, was bitten on the foot by a death adder through the stocking at 7.30 p.m. on April 5, 1927. She was brought fifty miles by ambulance and examined by Dr. D. Crawford seven and a half hours later. No symptoms were then present. Next day she complained of tenacious saliva which was difficult to expel, also of a sense of thickening of the tongue. Swallowing became difficult. The pulse rate varied from 96 to 132 and sweating and other features of vasomotor depression were evident. Later paralysis of the palate ensued and the speech was nasal in type. There was a sense of constriction of the chest, respiration became difficult, the face was flushed and the pupils dilated. On April 7, some thirty-six hours later, bilateral ptosis developed and neither food nor drink could be swallowed. By next day the patient had improved, the different symptoms and signs disappearing in much the same order as they came.

The immediate treatment carried out by the parents had consisted of ligature above the knee, deep incisions with a razor and the application of crystals of potassium permanganate to the wounds. Strychnine and pituitrin injections were subsequently given.

This case is of special interest as it shows the ascending nature of the bulbar paralysis. A long period intervening before the onset of symptoms generally indicates the inoculation of a sublethal dose of venom. In two sheep, however, injected with one certainly lethal dose of death adder venom experimentally the onset of paralytic symptoms was delayed for twenty-seven and a half hours in one and forty-four and two-third hours in another.

Brown Snake Bite.

Case IV.—Mrs. M.F.B., a snake charmer, aged twenty-nine years, was bitten through cord riding breeches just above the knee by a brown snake, which was stated to have been milked that morning. Two fang marks were present. A ligature was immediately applied at the middle of the thigh by her husband, the wound scarified and potassium permanganate was injected hypodermically. On admission to the hospital the temperature was 36·2° C. (97·2° F.), the pulse rate 102 and the respirations thirty-four per minute. Dr. F. Ceckie, who has supplied the following case history, saw the patient within a half of an hour of the bite and at this time her condition was excellent, though she was somewhat hysterical. There was a history of previous bites. Later vomiting and severe abdominal pain developed and vasomotor features with pallor, sweating, rapid weak pulse and low blood pressure appeared. Bright red blood was also passed *per rectum*. Bulbar paralysis characterized by difficulty in speaking and swallowing became established and the patient died of cardio-vascular failure twelve hours after being bitten.

This case presents several features of interest, perhaps the most important being the demonstration of the fact that, even with the brown snake which possesses short fangs with very limited powers of rotation, a lethal dose of venom was inoculated through material of considerable thickness. Milking had not removed the residual venom, but very few professional snake charmers have sufficient knowledge of the anatomical situation of the venom gland to do so. The passage of bright blood *per rectum* was not unusual. Persons after brown snake bite not infrequently show clinical evidence of internal hæmorrhage. Foxton's case[22] was of special interest as he found multiple hæmorrhages in the pons and medulla at autopsy. Hæmaturia and hæmatemesis were also present and despite immediate ligature, scarification down to the extensor tendons and the application of potassium permanganate crystals the patient died twenty hours after being bitten.

The Course of Snake Bite.

The course of snake bite is a variable one depending on the amount of poison injected and the presence or absence of certain complications. Martin[7] found experimentally that in black snake bite the cause of death was intravascular clotting and cardiac and respiratory failure, but that animals, especially dogs, might succumb at a later date from pathological changes in the lungs and kidneys.

In man bitten by the Australian colubrides death results from bulbar paralysis with respiratory and cardiac failure and if the patient survives three days, there is an excellent chance of recovery. Insufflation pneumonia may prove fatal after recovery from paralysis and rarely nephritic complications may ensue. Occasionally local sepsis and ulceration in the vicinity of the bite may prolong convalescence.

In fatal cases the interval between bite and death is subject to considerable variations and owing to its importance in respect to antivenene treatment the results of three independent series of observations on the death time in man are epitomized in Table VII.

TABLE VII.

The Death Time in Eighty-one Fatal Cases of Snake Bite in Australia.

Death Time.	Author.			Total.
	Tidswell.	Ferguson.	Fairley.	
1–6 hours ..	11	2	2	15
7–12 hours ..	8	2	4	14
13–18 hours ..	11	6	2	24
19–24 hours ..		4	1	
24 hours and over..	20	16	2	38
TOTAL ..	50	30	11	81

Eight of the first fifteen patients died within three hours of being bitten.

Only eight of eighty-one persons died in less than three hours, so that abundant time would have been available for the institution of specific serum therapy in the vast majority of patients.

Six of Tidswell's and eight of Ferguson's series died later than forty-eight hours after the bite. One of Ferguson's series succumbed after twelve days' illness. The longest period between bite and death was thirty-six hours in the present series, but one patient who recovered, suffered from severe paralytic symptoms for fourteen days. The history of this patient who was in the Rockhampton Hospital under the care of Dr. J. B. Gordon, is of considerable interest and is recorded below.

CASE V.—C.M., aged thirty-one years, a snake charmer, was admitted to hospital within three hours of being bitten by a tiger snake. An attempt had been made to scarify the wound and his own remedy applied.

On admission the patient was very collapsed and drowsy. There was an incision on the back of the forearm about five centimetres (two inches) below the elbow and about six millimetres (a quarter of an inch) below this there were two fang marks. The pupils were contracted and the temperature subnormal. Stimulants were administered and potassium permanganate injected locally.

Next day the patient was still drowsy and complained of severe general pain and subsequently generalized muscular weakness and paresis developed. The patient was quite unable to use his limbs. Ptosis and signs of bulbar paralysis such as difficulty in swallowing then appeared and this condition persisted for a period of fourteen days when he began gradually to recover power over his musculature. Improvement continued and the patient made a perfect recovery. He still handles snakes.

A paralysis of such delayed onset and duration is quite exceptional, though I have seen a somewhat similar condition in a sheep partially immunized to tiger snake venom. This animal stood eight certain lethal doses without symptoms, but succumbed six days after the injection of twelve lethal doses of venom. In this instance paralytic features did not develope until the third day. Many snake charmers acquire a certain grade of immunity from previous bites and probably such a condition explains the atypical clinical course recorded above.

PROGNOSIS.

Time will not permit a detailed discussion of this question, but information regarding the species of snake, its potential venom-producing capacity as indicated by the distance between the fang punctures, the number of fang marks present, the intervention of clothing over the bitten area and the age and body weight of the patient should all be ascertained. Hæmaturia, hæmoptysis, hæmatemesis and other signs of internal hæmorrhage indicate a bad prognosis, while the early onset of paralysis especially involving the bulbar nuclei has a similar significance.

An interfang measurement under one centimetre probably indicates a small dose and one over one and a half centimetres a large dose of inoculated venom. This appears to hold with all species, for though the fangs in even large brown snakes may not be more than 1·1 centimetres apart, they are all poor venom producers.

TREATMENT.

Much of the early literature of snake bite in Australia revolved around the value of certain remedial measures, such as the intravenous injection of ammonia introduced by Halford[17] and the intensive strychnine treatment advocated by Mueller.[24] Neither mode of therapy has stood the test of time, but it should be noted that while Mueller considered strychnine to be a physiological antidote, Halford utilized ammonia purely for its stimulant effects. Croll[25] has rightly emphasized the dangers of overdosage with strychnine in snake bite poisoning.

At the present time only two methods of treatment are recognized as being of value after the inoculation of a lethal dose of venom. These consist of antivenene and certain local measures such as ligature, incision, excision and the injection of certain chemicals like chloride of gold (1%), potassium permanganate (1%) and calcium hypochlorite.

Local Treatment.

The principle underlying all forms of local treatment is to remove, destroy or render inert venom inoculated into the tissues before the absorption of a lethal dose into the systemic circulation.

What is not generally realized is the extreme shortness of this absorption period, when several lethal doses of venom have been injected into the subcutaneous tissues, as is frequently the case with natural tiger snake and death adder bites.

Experiments on sheep made during the present investigation have shown that the absorption time is less than two minutes under conditions of natural

bite by *Notechis scutatus*. In these animals it is essential to bring the circulation to a standstill by ligature well within the two-minute period, if any local measures are to be effective, and there is no reason to suppose that the position in regard to man is different.

Ligature.

Ligature should be applied around a single bone on the heart side of the bite, the thigh and the arm being the sites in foot or hand bites and never the leg or forearm. In addition it may be placed near the base of the implicated digit in a toe or finger bite. Continuous or intermittent ligature may be practised, this question being taken up in more detail in the separate paper dealing with this subject. Complete cessation of the circulation must be produced and in all our animal experiments an incision was made into the ligatured area to make certain that this had been attained.

It is generally agreed that the efficacy of ligature as a life saving device is dependent on the thrombase content of the venom inoculated and that in the absence of this constituent all it does is to hold up the poison temporarily in the ligated limb. This is its recognized action in colubride snake bite.

It has been seen that in the case of the death adder and the copper-head, thrombase is absent and in bites by these snakes ligature *per se* cannot be regarded as doing more than prolonging the death time.

Tiger snake and black snake venom are exceptional in containing considerable amounts of thrombase and Martin[7] showed experimentally in rabbits that immediate ligature saved life even when as much as six lethal doses of the latter venom were injected. This result was found to be due to the effect of thrombase in causing local clotting of tissue fluids and in adjacent vessels under conditions of circulatory stasis, thus leading to permanent localization and destruction of the venom *in situ*. Tidswell's series[1] of seventy-five cases of bite by potentially lethal snakes showed that fourteen out of fifty-five persons to whom ligatures were applied, died (25·4%), whereas only six out of sixteen to whom no ligatures were applied survived (55% mortality). To be comparable, however, the mortality rate in ligatured and non-ligatured series must be determined for each species of snake and in the absence of such information no conclusions of significance can be reached.

In the literature there are many examples of patients bitten by the tiger snake, death adder and brown snake who died despite ligature, while in connexion with persons who recovered after ligature, the time and mode of application of the ligature are such that this procedure could not possibly have influenced the result.

During the present investigation a large series of experiments has been carried out on sheep injected subcutaneously with tiger snake venom, but ligature has consistently failed to save these animals even from one certain lethal dose.

This result was attributed to the decreased sensitiveness of the sheep to thrombase and as rabbits are known to be highly sensitive, the question naturally arises regarding the sensitivity of human tissues to this substance. Deaths from intravascular thrombosis are not recorded in man, though they may occur in sheep naturally bitten by tiger snakes. This certainly suggests that man is not unduly sensitive to thrombase and in consequence that no more can be expected from ligature than prolongation of the death time.

That prolongation of the death time does occur I have on several occasions shown by ligaturing the limb of sheep effectively and injecting pure fresh tiger snake venom (0·13 cubic centimetre or fifty-five to seventy certain lethal doses) into the subcutaneous tissues of the ligatured area, care being taken to avoid injection into a vein. The ligature was left *in situ* for periods of from four to six hours, during which no symptoms developed. After its removal the animals invariably became paralysed (forty to ninety minutes) and died about two hours later, the maximum intervening period being two hours and ten minutes. Autopsy revealed typical findings with multiple hæmorrhages and congestion of the viscera. Intravascular thrombosis was not present.

Brazil[26] states that systemic absorption of poison may result by direct tissue spread even in the presence of effective circulatory stasis induced by ligature, but this has not been borne out by the present experimental results. General absorption proceeds with such rapidity that it is essential in experiments of this nature to make the injection into an area where the circulation has already ceased.

Incision.

Incision or scarification is generally combined with ligature in the treatment of snake bite, but despite its general use it is not a procedure which *per se* materially assists in the removal of venom. It, however, does afford an excellent index to circulatory stasis, the reappearance of bleeding indicating that the ligature is slipping. Perhaps this is its most important function. It may also reveal the presence of broken fangs in the fang punctures and permits the application of suction either by the mouth, a breast pump or a Bier's suction glass. As Ferguson[2] showed, it is essential to wash off any venom deposited on the skin itself, as otherwise incision and scarification may lead to increased absorption.

Venom is a colloidal solution which is injected under pressure infiltrating the tissue spaces surrounding the fangs. These often enter the tissues obliquely as during erection the fangs frequently point outwards and subsequently they are retracted backwards and somewhat inwards, poison being deposited throughout their course. Under these circumstances drainage of the fang track by a straight incision backwards is obviously impossible, while an interfang incision is also useless in the colubrine snakes at least.

The following experiment indicates the failure of this treatment in sheep:

Three animals weighing 42, 35 and 43 kilograms were utilized. A tiger snake was allowed to bite a shaved area of skin over the distal end of the metacarpal bone of the foreleg, a rubber tourniquet being immediately tightly applied on the heart side over this bone. Two incisions twelve and a half millimetres long were made as soon as possible through the skin and subcutaneous tissues down to the tendons. In the first animal (number 140) the ligature was removed for five seconds at the end of twenty minutes and immediately reapplied. Within forty minutes paralysis had developed and seven minutes later the animal died with the ligature *in situ*. In the case of the other two animals (numbers 172 and 168) a 5% solution of potassium permanganate was poured over the skin and into the incisions, and continuous ligature for periods of forty-five and fifty-five minutes was adopted. Paralysis developed in one hour fifteen minutes and three hours thirty-five minutes respectively and death ensued in one hour thirty minutes and four hours fifty-three minutes. The snakes used were of average size varying from ninety-five to one hundred and four centimetres in length, while the interfang measurement lay between 1·35 and 1·6 centimetres. In each snake subsequent milking showed the presence of residual venom.

These experiments on large sized animals illustrate the failure of the standard treatment advocated and practised in Australia. The conditions entirely favoured the animal bitten, for the whole operation was completed within one and a half minutes of receipt of the bite. In the field such rapid treatment becomes impossible and these results can be interpreted only as indicating a corresponding grade of ineffectiveness in man.

Excision.

Owing to the difficulty of ascertaining the course taken by the fangs and also on account of the drag and distortion of the tissues during bite immediate ligature followed by excision of the whole area is the most rational form of local treatment at present available. Excision of the skin and subcutaneous tissue over an area of 3·2 by 1·9 centimetres with the longest diameter through the fang punctures should remove the infiltrated tissue in the case of all the Australian snakes, but on account of the greater length and distance between the fangs a more extensive and deeper excision may be necessary in the case of the death adder. Owing to compression of the tissues poison may be more deeply deposited than measurements of the fangs indicate and in hand and foot bites the dissection should always be made down to the bone in the case of the tiger snake as well as the death adder. Swabbing the excised area with a 5% solution of potassium permanganate and the application of an Esmarch's bandage from the ligature downwards may be of value and the ligature should be kept on for a period of twenty to thirty minutes following the operation.

Martin and Lamb[23] and others recommend excision, but Acton and Knowles[27] did not think it could be effective in colubride bites owing to the rapidity with which venom is absorbed into the blood stream.

The following experiments indicate its value in sheep naturally bitten by *Notechis scutatus*, the failure or success of the procedure being dependent on the rapidity with which an efficient ligature is applied.

Four sheep (numbers 166, 178, 180 and 181) weighing from thirty-two to forty-five kilograms were used in this experiment. The forelimbs were shaved and a tiger snake was allowed to bite the soft tissues covering the lower end of the right metacarpal bone.

In every instance the snake was observed to bite effectively and residual venom was subsequently demonstrated by milking. The snakes utilized were of average size, their length varying from ninety-one to one hundred and one centimetres, while the interfang measurements were 1·55, 1·3, 1·7 and 1·6 centimetres respectively.

A broad rubber tourniquet (1·5 centimetres) was stretched, wound round the upper half of the metacarpal bone and knotted. This was done immediately after the bite except in one animal (number 180), when some unavoidable delay occurred. The skin and subcutaneous tissues were next rapidly excised, a square area measuring about 2·9 by 2·9 centimetres being dissected away as rapidly as possible. A strong solution of permanganate of potassium was poured over the excised area and the ligature was left *in situ* for periods of forty-five, thirty, one hundred and sixty-five minutes respectively. Profuse bleeding occurred in every instance on its removal.

Apart from the local condition no systemic symptoms developed except in the case of sheep number 180, where the application of ligature had been delayed for a period of approximately two minutes. This animal became completely paralysed in three hours ten minutes and died three hours and twenty-four minutes after being bitten. At autopsy the usual visceral congestion and hæmorrhages were seen.

Commentary.

These results again illustrate in a striking fashion the urgency for the immediate application of ligature following natural bite; when this is done, excision as described above proves to be a life-saving measure.

The absorption time of a lethal dose of venom naturally varies with the quantity inoculated and in the case of different species of animals according to natural resistance and body weight. It is thus possible that the absorption time under conditions of natural bite in sheep (about two minutes) is less than the corresponding period for man, but when large doses of venom are injected no great extension of the time is probable. Unfortunately, owing to the practical difficulties existing under field conditions it is a mode of treatment which will rarely be effectively accomplished.

Amputation comes in a similar category. It is only justified in finger and toe bites, when immediate effective ligature had been applied and possesses no advantages over a wide and deep excision of the bitten area. Local treatment in areas not capable of ligature is most unsatisfactory and in sheep bitten by *Notechis scutatus* widespread excision extending down the muscle and the application of a large Bier's suction pump failed to save life when the excision was commenced within two minutes after receipt of the bite. When a small quantity of venom (two minimum lethal doses) was injected, however, excision within a period of five minutes was successful.

Chemical Agents.

Little work has yet been done on the different group of drugs which like thrombase cause local

thrombosis in injected areas. Chemical agents like gold chloride, potassium permanganate and hypochlorite of lime which destroy venom *in vitro*, have, however, been widely utilized.

The usual method of packing Condy's crystals into incisions through the fang puncture was shown by Acton and Knowles[27] to be of no practical value in the case of cobra venom and they advocated abandoning the use of the Lauder Brunton lancet altogether. Potassium permanganate in solution may be beneficially used for flushing out excised areas and especially for destroying venom deposited on the skin. As an alternative to incision a 1% to 5% solution may be injected into the fang punctures and the surrounding tissues, but Acton and Knowles[27] have shown that ten to twenty cubic centimetres of a 1% to 5% solution of chloride of gold is a more efficient remedy. Unfortunately both these substances cause necrosis and gangrene of the tissues and are far more efficient with viperine than with colubride venoms. They are, therefore, not likely to prove effective in bites by venomous snakes in Australia.

General Management.

Apart from stimulant treatment, warmth and rest, care must be taken to keep the mouth and nasopharynx as free from mucus as possible, and also to prevent the entry of vomitus into the larynx when bulbar paralysis develops. For this reason when the patient does not vomit early, it may be advisable to remove the gastric contents with a stomach pump, and subsequently to give all fluids and nutrients *per rectum*. Artificial respiration is a measure which may temporarily prolong life. Its greatest value is in increasing the time available for the administration of antivenene. Alcohol, ammonia and strychnine have had a wide vogue as stimulants and for vasomotor failure adrenalin, pituitrin and bandaging of the limbs are advocated. Such measures may be helpful, but the only satisfactory treatment is the one already established in the United States, South America, India and other countries, namely specific antivenene therapy.

The Case for Antivenene Treatment.

Antivenene has the great advantage over all other remedies in that it definitely saves life even after many lethal doses of venom have entered the circulation and paralysis is well advanced.

Time will not permit a reviewing of the work of Calmette[28] [29] regarding the universality of the curative properties of his *serum antivenimeux* nor those fascinating studies in immunology by which the views of the French school were shown to be erroneous.

C. J. Martin[30] soon demonstrated that Calmette's antivenene lacked curative properties as far as tiger snake venom was concerned and later Tidswell[1] found it to be equally useless against brown snake, black snake and death adder venoms. We now know that each antivenene is antidotal only against the particular venom employed in its production and that Calmette's serum was chiefly effective against cobra venom.

Martin[31] also proved that antivenene to be effective must be given intravenously, ten to twenty times as much being necessary if subcutaneous dosage is to yield an equivalent protective effect.

Tidswell[1] produced an antivenene against *Notechis scutatus* by immunization of the horse. Injections were commenced in 1898 and continued over a period of three and a half years. Its protective action was shown by the fact that rabbits receiving one minimum lethal dose of venom were saved, provided one cubic centimetre of antivenene per kilogram of body weight was injected subcutaneously or 0·1 cubic centimetre intravenously within one hour. Such an antiserum would not be sufficiently potent for more severe tiger snake bites in man and chemical concentration would be necessary. Unfortunately, as Caius and Iyenger[32] have shown, reduction in volume is not necessarily associated with a corresponding increase in potency as far as cobra venom is concerned and this is one reason why high titre antivenenes are essential.

Our chief killing snakes are the tiger snake and the death adder and owing to their different geographical distribution these antivenenes could be put up in monovalent form. The interval between death and bite has been shown to be adequate for the administration of antivenene, which is also known to keep well even under tropical conditions. Caius and Anderson[33] in India demonstrated that the initial depreciation occurring during the first six months was subsequently followed by a rise of titre so that the serum in the course of twelve to fourteen months might increase in potency to its original titre. Light and heat in an ordinary room in the tropics exercise no particular influence on its potency. Finally in America Crimmins[34] has shown what remarkable results can be achieved by a modern organized effort. Twenty-one out of twenty-one persons bitten by rattle snakes and treated with antivenene recovered, while thirteen out of thirty untreated died. Where the patient could not be brought in from the country, antivenene was sent out by aeroplane, a total of 2,600 miles being covered by air in hurrying serum to victims of snake bite. Conditions in America appear to be very similar to those encountered in this country and their methods of handling the ophidian problem might be followed with considerable benefit.

Prophylaxis.

No dissertation on snake bite would be complete without reference to methods of prevention.

One hundred and fifty-four out of two hundred and eighty-one persons were bitten below the knee and the vast majority of these could have been prevented by the use of boots and leggings or putties. The fangs of any of the Australian colubrides can pierce one layer of material satisfactorily and a snap bite by a tiger snake on a sheep whose foreleg was covered by a thick woollen stocking proved fatal within one hour and twenty-five

minutes. Immediately the snake struck the sheep pulled its foot away and the whole bite only occupied a fraction of a second.

When the same sock was employed covered by two layers of a regulation army putty, natural bites in the case of both the tiger snake and the death adder proved ineffective, the poison being injected into the sock and not into the skin which showed no fang punctures. The death adder employed in this experiment had average sized fangs and showed an interfang measurement of 1·75 centimetres. Despite this finding, however, leggings are in my opinion preferable to putties in country where adders abound. In a natural bite the fangs of the death adder were found to penetrate through the side of an ordinary shoe, but one of the fangs was lost in the process and an effective bite was improbable. With thick boot leather penetration becomes impossible.

Snakes bite man by accident more often than by design and though both brown and tiger snakes enter houses, this is not a common event. Death adders are found on paths and roads at night and it is very advisable to carry a lantern or torch in death adder country.

Tiger snakes and brown snakes frequent logs and copper-head and black snakes rabbit burrows. The common practice of thrusting the hand into such places is to be deprecated and only a month ago one youth was fatally bitten on the face while crawling into a hollow log after a rabbit. Attention to the natural habits of the ophidia and common sense would prevent many bites.

Finally legislation preventing the unauthorized sale of so-called specific antidotes for snake bite and the handling of venomous snakes in side shows is required. The expectancy of life in the snake charmer is very low and the sale of his futile remedies is dangerous to the public. This question is one concerning which the various Branches of the British Medical Association in Australia might well take coordinated action.

Summary and Conclusions.

1. The mode of action and the clinical and pathological effects produced by different Australian colubride venoms in sheep are briefly recorded.

2. Immediate ligature combined with incision and the application of potassium permanganate solution failed to save life in sheep naturally bitten by *Notechis scutatus*.

3. In sheep excision of the bitten area proved an effective therapeutic procedure only when ligature was applied immediately after the bite, that is within one minute.

4. The necessity for the establishment of antivene treatment in the case of tiger snake and death adder bites in Australia is emphasized. At present no satisfactory treatment is available and once a lethal dose of venom is absorbed, the patient must die.

5. Certain prophylactic measures are discussed. These include the use of leggings and putties in snake country and legislation directed to prevent the sale of patent antidotes and the handling of venomous snakes in side shows.

Acknowledgements.

I wish to express my thanks to the various practitioners who have replied to the *questionnaire* circulated from the Institute during the past twelve months, also to Professor Cleland for supplying me with a bibliography of the recorded cases of Australian snake bite in man. Dr. J. H. L. Cumpston supplied the tables on deaths in the Commonwealth from venomous bites and stings (1910-1926) and in this and other ways has assisted us in our investigations.

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THE DENTITION AND BITING MECHANISM OF AUSTRALIAN SNAKES.[1]

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NON-POISONOUS as well as the poisonous snakes are carnivorous and the characteristic difference of the former group lies rather in the absence of anteriorly situated, deeply grooved or canalized fangs for efficient inoculation purposes than in the presence of a non-venomous saliva. The existence of poison glands in *Tropidonotus natrix* and other non-venomous snakes has long been recognized and Alcock and Rogers[1] showed that an infusion of the parotid glands of some of the so-called non-poisonous colubrides (*Aglypha* and *Opisthoglypha*) killed small mammals with symptoms resembling those of cobra poisoning when injected subcutaneously. The sera of these snakes as well as extracts of their harderian glands were found to be harmless, so that the venomous nature of the salivary secretion in the ophidia appears to be one of degree rather than of kind.

Primarily snake venom is a digestive juice, its kinases activating pancreatic secretion, but in the poisonous snakes it subserves another function, namely that of a defensive or offensive secretion whereby its prey is secured.

THE POISON GLANDS.

Noguchi[2] has pointed out that the poison gland of the fangless snakes is of mixed type consisting of an anterior greyish-red portion composed of mucous glands with numerous ducts and a posterior greyish-white portion, provided with only one duct, which histologically is of the nature of a serous gland. This latter is the homologue of the parotid gland of the mammalia and is of special biological significance, since it is the first occasion in which a serous glands appears in the amphibia.

In the proteroglyphous colubrides of which the common venomous Australian snakes are all examples, the serous and mucous glands are distinctly separated. The poison gland proper (parotid) is superficially situated on the side of the head behind the orbit and above the upper lip corresponding in the case of the death adder to the posterior portion of the fourth, the fifth and the anterior aspect of the sixth supralabial as well as to the apex of the lowest temporal scale (see Figure I). The supralabial mucous glands are situated more anteriorly and pour their secretions into the laterally compressed venom duct as it passes forward in its course along the margin of the upper lip (see Figure IX) to open by a papilla on the anterior wall of the *vagina dentis*.

The poison gland is surrounded by a thick fibrous tissue capsule to which are attached fascial processes suspending the gland in position and into which the fibres of the anterior temporal muscle (mandibular) are inserted. Photomicrographs of sections through the venom glands of the tiger snake, death adder and brown snake (see Figures VI, VII and VIII) show very clearly the intimate relationship existing between this muscle, the gland capsule and the gland itself. The latter is of compound racemose type consisting of an interalveolar framework of connective tissue and alveoli lined with short columnar epithelium in whose capacious lumina the venomous secretion is stored.

A section through the corresponding retro-ocular maxillary salivary gland of the carpet snake is shown for purposes of comparison (see Figure X).

Weir Mitchell[3] weighed the venom glands of a series of rattlesnakes and found little difference between the size and weight of the snake and the weight of the gland beyond a general increase in the size of the organ with that of the snake. This has not been done with the Australian colubrides, but direct measurement during the present investi-

[1] This research was carried out under a grant from the Commonwealth Government Department of Health.

gation has shown that in the case of the brown snake the venom gland is both relatively and absolutely smaller than in the other species, a finding which is probably related to the general narrowness of its skull.

In the broad headed death adder the venom gland reaches its largest dimensions, its size in the other snakes being intermediary.

OSTEOLOGICAL FEATURES OF THE SKULL.

The dentition and osteological features of the ophidian skull have undergone a series of modifications and adaptations determined by their habits of feeding on large sized prey. The various bones entering into the formation of the roof and floor of the mouth present numerous, strongly recurved teeth, while the rami of the lower jaw are joined only by an elastic ligament. The upper jaw and palate are also independently movable. In addition the lower jaws are more widely separated from the base of the skull by the quadrate bone which articulates with the squamosal on the one hand and the mandible on the other (see Figures XII to XXIII). Finally the mobile trachea is located well forward on the floor of the mouth, enabling breathing to be continued through the whole act of swallowing (see Figure III).

Phisalix[4] in her classical work on the ophidia has emphasized the anatomical and functional independence of the pterygo-palatine-transverse arches in the non-poisonous constricting snakes, as well as in the colubrides and the vipers and has also traced certain important osteological transitions through the different families commencing with the *Boïdæ*, passing through the aglyphous, opisthoglyphous and proteroglyphous *Colubridæ* and reaching their highest evolutionary development in the *Viperidæ*. Briefly these changes consist of a distinct shortening of the skull and of the superior maxillæ, elongation of the quadrate bones and in the case of the vipers in a capacity to rotate the maxillæ on their transverse axes of articulation with the prefrontals producing elevation of the fangs. Phisalix also points out that increase in the length of the quadrate bones permits greater depression of the mandible with maximum dilatation of the mouth, while the shortening of the maxillæ entails the interdependence of the two pterygo-palatine-transverse arches, enabling the species to acquire the faculty of using the fangs alternately and so of not relaxing its hold on its prey when once seized. The forward projection of the rest of the mobile palate is rendered possible by the same mechanism, while the lower jaws also possess independent power of movement.

The appearance of grooved and canaliculated fangs for the inoculation of venomous saliva has obviously occurred as a late evolutionary modification of the ordinary biting apparatus possessed by non-poisonous snakes. In order to appreciate adequately the real mechanism of snake bite, the cranial osteology of both must be understood. The two non-poisonous snakes studied in the present investigation were the diamond snake (*Python spilotes*) and the carpet snake (*Python spilotes* var. *variegatus*) belonging to the *Boïdæ*. Both possess elongated skulls with short strong quadrate bones and long superior maxillæ carrying numerous recurved teeth (see Figures XII, XIII and XXIIIb).

In *Python spilotes* the distance from the premaxilla to the anterior border of the orbit is about twice the diameter of the orbit itself (see Figures XII and XIII), whereas in the Australian poisonous snakes the two distances are approximately equal. This anterior shortening of the skull is due to decrease in the size of the prefrontal, nasal and maxillary bones, while the posterior shortening which is also very evident, is attributable to a decrease in the length of the parietals.

The Pterygo-palatine-transverse Arch.

In the non-poisonous snakes the superior maxilla consists of an elongated bone extending along the upper lip to behind the orbit. In the carpet and diamond snakes it is somewhat incurved and rounded at its anterior extremity where it makes ligamentous connections with the pre-maxilla, while its postero-superior surface articulates obliquely with the short ectopterygoid (see Figures XII and XIII). Near the middle of the bone its internal border bulges inwards impinging on the posterior aspect of the external surface of the palatine bone. The endopterygoid and palatine bones are also strong and well developed (see Figure XXIIIb). Definite but limited forward movements of the pterygo-palatine-transverse arch were demonstrated by electrical stimulation of the spheno-pterygoid muscle in living specimens in which the brain had been destroyed.

Five distinct sets of recurved teeth are found in the roof of the mouth, the central premaxillary and the two lateral maxillary constituting a continuous outer marginal line shaped like a U, while the double row of pterygo-palatines form two straight inner lines which are readily demonstrated in dental impressions of the bite taken during life (see Figures XXXIV and XXXV).

In the Australian poisonous colubrides the maxillæ are shortened to a variable degree, but never to the extent met with in the vipers in which post-maxillary teeth are absent.

The superior maxilla consists of an enlarged anterior portion which is thickened and rounded in front and thinned and concave behind, while its postero-external angle is prolonged backwards for a variable distance as a posterior arm bearing on its inferior surface from one to nine recurved post-maxillary teeth, the number varying in different species (see Figures XII to XXIII). A simple grooved fang or two transversely arranged fangs are found ankylosed to the broadened inferior surface of the maxilla anteriorly.

The superior surface of the maxilla which faces upwards and slightly backwards, articulates anteriorly with the inferior surface of the pre-

frontal which looks downwards and slightly forwards. This surface is generally smooth and flat, but in the death adder it may be somewhat concave, a structural modification which probably permits a further degree of rotation taking place. The anterior and posterior prefronto-maxillary ligaments are loose but well developed. They allow a considerable range of sliding movement at this joint and also act as check ligaments when these movements tend to be excessive. The internal border of the anterior expanded portion of the maxilla forms ligamentous attachments to the external and superior surface of the palatine bone so further increasing the stability of the palatine arch. At rest the inferior surface of the maxilla is horizontal and the recurved fangs point backwards at an angle of about 40° to 50°. The posterior arm articulates obliquely with the broadened anterior end of the ectopterygoid which in turn ankyloses with the endopterygoid somewhat in front of the junction of its posterior and middle third. The endopterygoid is connected with the palatine bone anteriorly, while its posterior wing is joined to the quadrato-mandibular joint by muscular and ligamentous connexion (see Figure XXIIIa).

In the Australian colubrides each pterygo-palatine-transverse arch acts as a single entity and when the protractor muscles of the palate draw the endopterygoid forward, they inevitably bring with it the palatine bone and the ecto-pterygoid which impinging on the posterior arm of the maxilla drives it forwards and upwards on its prefrontal articulating surface. This produces a variable grade of elevation and forward rotation of the fangs, the extent of which is accurately indicated by the angle formed at the ectopterygoid-maxillary junction.

The Maxillary Shortening Observed in Different Species.

Since shortening of the whole skull in these poisonous snakes occurs with such uniformity, it becomes a matter of importance in considering the dimensions of the superior maxilla in different species to determine whether the latter bone is simply sharing in the general longitudinal compression of the cranium or whether it is undergoing an excessive decrease in length relative to the skull as a whole.

If the length of the skull in centimetres as measured from the anterior border of the premaxilla to the posterior border of the occiput be divided by the length of the superior maxilla itself a figure, which for brevity I have named the maxillary index, is obtained affording an accurate basis for comparison of the maxillary shortening in different species of ophidia.

The average results obtained from skull measurement on the present series are epitomized in the accompanying Table I. It will be seen that the maxillary index is least in the non-poisonous snakes (1·5 to 1·57), that it steadily increases with the brown snake (2·65), the black snake (3·28), the copper-head (3·4) and the tiger snake (3·56), until it reaches its maximum with the death adder (3·72). In the case of the vipers it would be greater still.

Running parallel to this shortening of the maxilla there is a corresponding decrease in the average number of maxillary teeth which vary from 15·5 in the diamond to 1·7 in the death adder, the fang in the case of the poisonous snakes being excluded from the count.

The brown snake approximated most closely to the non-poisonous species, the average number of its post-maxillary teeth being 7·25, while for the copper-head, black snake and tiger snake it was 4·5, 4·0 and 3·0 respectively.

As a compensatory measure for shortening of the maxilla progressive elongation of the ecto-pterygoid was also noted throughout the series, being longest in the death adder and tiger snake and shortest in the brown and the non-poisonous snakes.

The natural forward movement of the pterygo-palatine-transverse arch was studied in living snakes, also experimentally by electrical stimulation of the protractor and retractor muscles in specimens in which the brain had been destroyed. It was then found that a definite relationship existed between the amount of forward movement and the maxillary index, being greatest in the case of the death adder and much decreased in the brown, the carpet and the diamond snake. The copper-head, black and tiger snakes possessed a moderate degree of independent protraction of the palatal arches.

Thus, in the five common poisonous snakes of Australia a very definite relationship is traceable between maxillary shortening and decreasing maxillary teeth on the one hand and the forward projection of the palatal arch on the other.

The Elevation of the Fangs.

A definite degree of rotation of the maxilla with elevation of the fangs is essentially a viperine characteristic and it has rarely been reported in the case of colubride snakes.

Boulenger,[5] however, noted its occurrence in the non-poisonous colubrid, *Xenodon merremii*, which belongs to the series *Aglypha* and possesses a vertically movable maxillary bone carrying large posterior teeth and smaller anterior recurved ones. The range of rotation was here quite comparable to that observed in the vipers. Phisalix[4] also points out that the transition from the vipers or *Solenoglyphæ* to the *Proteroglyphæ* is shown in the genus *Dendraspis* (*Dendraspis angusticeps*), in which the maxilla, although tolerably long, is able to rotate under the slight pressure of the ecto-pterygoid and to project its fangs forward during biting. During the present investigation special attention has been paid to the degree of elevation and rotation of the fangs obtaining in the different colubride snakes under review.

Skulls were prepared with the pterygo-palatine-transverse arch in its usual resting position, partially drawn forward and completely protracted. By such means it was possible to study at leisure the exact modifications which the cranial bones and articulating surfaces must undergo as a result of

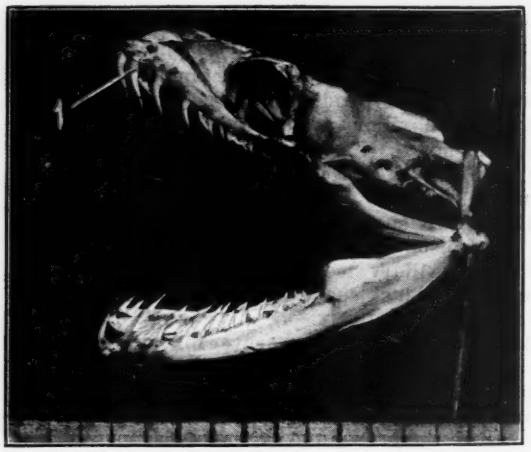

FIGURE XII.
Carpet Snake.

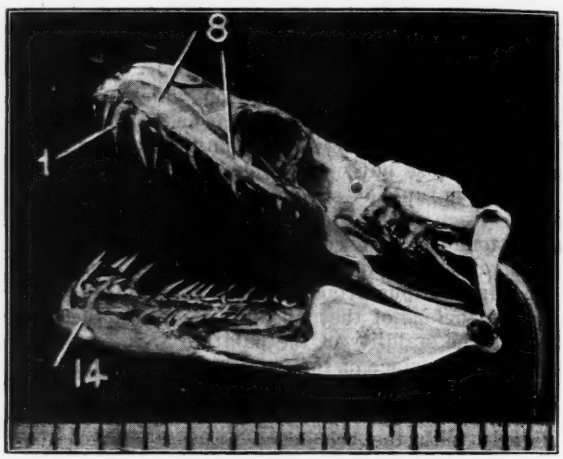

FIGURE XIII.
Diamond Snake.

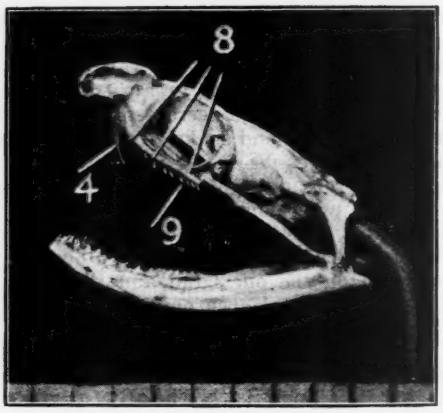

FIGURE XIV.
Brown Snake.

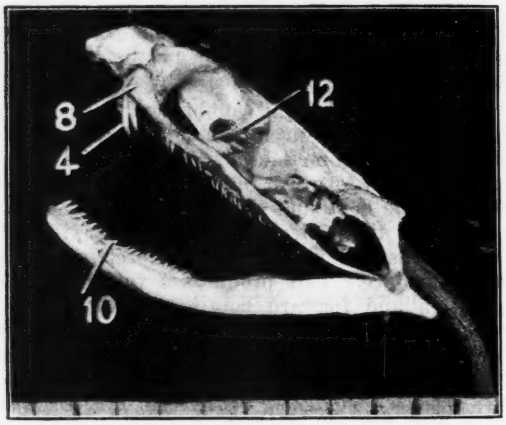

FIGURE XV.
Copper-head Snake.

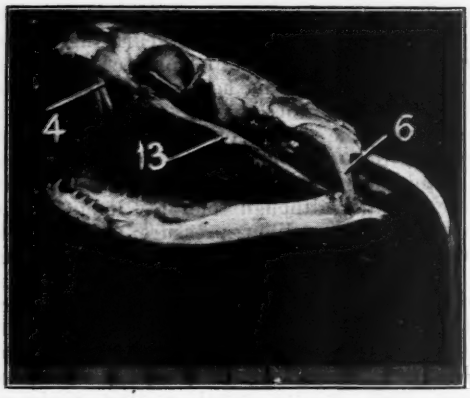

FIGURE XVI.
Speckled Black Snake (*Pseudechis guttatus*).

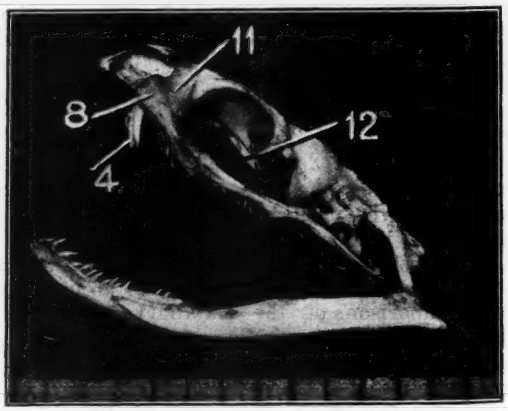

FIGURE XVII.
Black Snake (Maximum Rotation of Fangs).

SKULLS OF AUSTRALIAN SNAKES.

Note the large number of maxillary teeth in the case of the non-poisonous carpet and diamond snakes. In all the poisonous snakes the number of the post-maxillary teeth is limited and the maxilla itself is shortened. They are most numerous in the brown snake (Figure XIV).

1. Maxillary Teeth. 4. Fang. 6. Quadrate. 8. Maxilla. 9. Post-maxillary Teeth.
10. Mandibular Teeth. 11. Prefrontal. 12. Post-orbital Process. 13. Ecto-pterygoid. 14. Mandible.

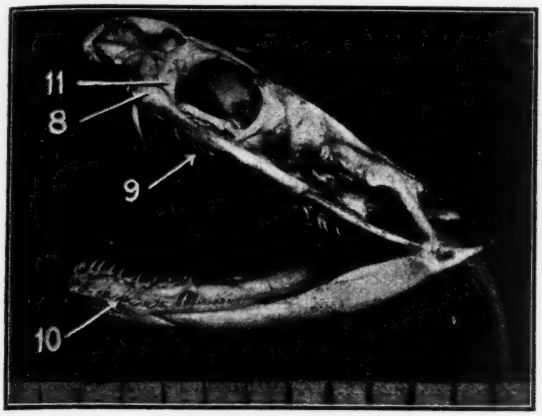

FIGURE XVIII.
Tiger Snake (Resting Position of Fangs).

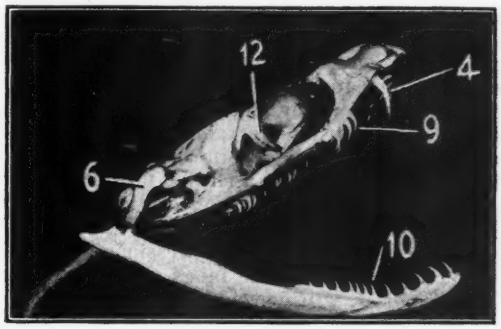

FIGURE XIX.
Tiger Snake (Maximum Rotation of Fangs).

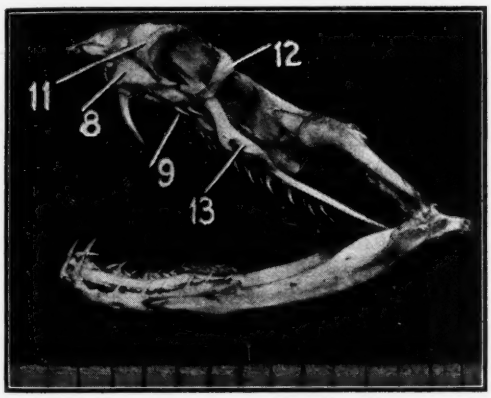

FIGURE XX.
Death Adder (Resting Position of Fangs).

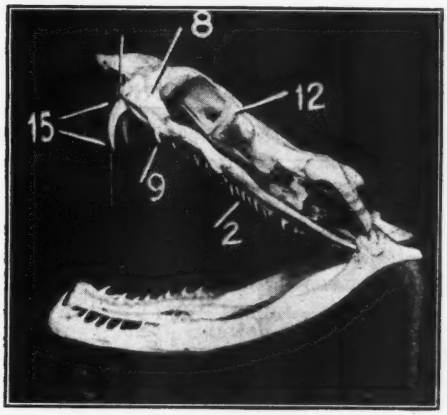

FIGURE XXI.
Death Adder (Mid-rotation of Fangs).

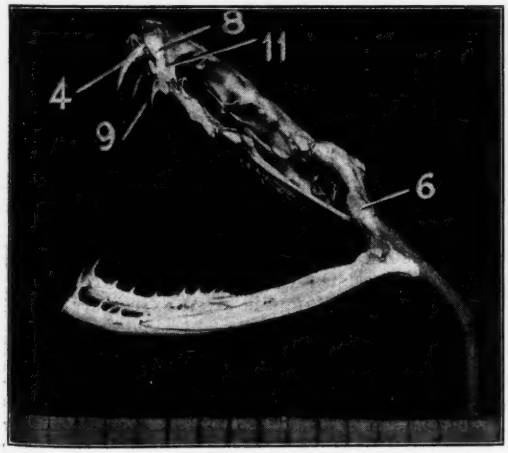

FIGURE XXII.
Death Adder (Maximum Rotation of Fangs).

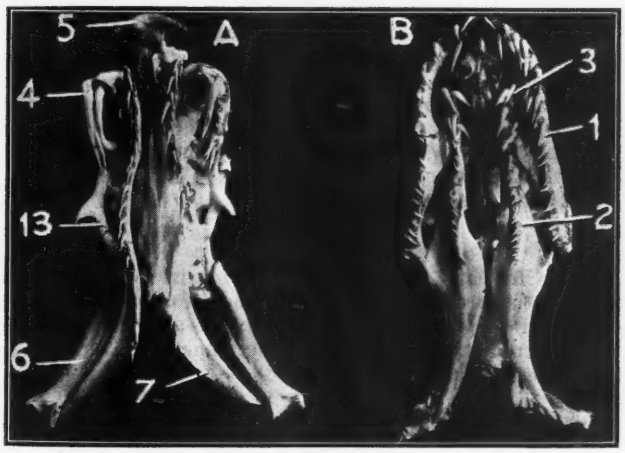

FIGURE XXIII.
(*a*) Death Adder, (*b*) Carpet Snake (Roof of Mouth).

SKULLS OF AUSTRALIAN SNAKES.

1. Maxillary Teeth. 2. Pterygoid Teeth. 8. Maxilla. 4. Fang. 5. Premaxilla.
6. Quadrate. 7. Posterior Wing of Sphenoid. 3. Palatine Teeth. 9. Post-maxillary Teeth. 10. Mandibular Teeth.
11. Prefrontal. 12. Post-orbital Process. 13. Ecto-pterygoid. 15. Venom Groove.

the stress and strain of the forward drag of the protractor muscles on the palatal arch during bite. The results were confirmed during the collection of venom by repeated observations on the mechanism of bite, as well as by electrical stimulation of the spheno- and parieto-pterygoid muscles in living snakes in which the brain had been destroyed. Finally, by means of a series of plaster and amalgam casts the actual position of the fangs during maximal protraction of the palatine arch was reconstructed from dental impressions of the bite.

The angle at which the fangs of the snake enter a bitten object, will depend not only on the degree of maxillary elevation, but also on the extent to which the mouth is open, and the obliquity of the upper jaw relative to the bitten surface.

In all these colubrides the fangs are much recurved, so that the angle which they actually subtend with the margin of the upper jaw, is difficult to determine. For purposes of estimation the fang line has been taken as a continuation of the line joining the posterior border of the base of the fang to its apex, while the margin of the jaw has been taken as the continuation of the line of the ectopterygoid. Based on these calculations the maximum angle of rotation through which the fangs pass is approximately 40° to 50° for the death adder, 30° to 35° for the tiger snake and 25° to 30° for the copper-head and black snakes. In the brown snake it does not appear to exceed 10° to 15°.

TABLE I.

The Maxillary Index and Forward Movement of the Pterygo-palatine-transverse Arch in Certain Non-poisonous and Poisonous Australian Snakes.

Species of Snake.	Average Measurements in Centimetres.		The Average Maxillary Index.[1]	Average Number of Maxillary Teeth (Fangs excluded).	Forward Movement of Pterygoid Arch.	Degree of Elevation of the Fangs.
	Length of Skull.	Length of Maxilla.				
Diamond snake (*Python spilotes*)	4·5	3·0	1·5	15·5	Limited	—
Carpet snake (*P. spilotes* var. *variegatus*)	5·9	3·75	1·57	14·0	Limited	—
Brown snake (*Diemenia textilis*)	3·0	1·13	2·65	7·25	Limited	Slight (10° to 15°)
Black snake (*Pseudechis porphyriacus*)	4·1	1·25	3·28	4·0	Moderate	Moderate (25° to 30°)
Copper-head (*Denisonia superba*)	3·4	1·0	3·4	4·5	Moderate	Moderate (25° to 30°)
Tiger snake (*Notechis scutatus*)..	3·1	0·87	3·56	3·0	Moderate	Moderate (30° to 35°)
Death adder (*Ancanthophis antarcticus*)	3·5	0·94	3·72	1·7	Considerable	Considerable (40° to 50°)

[1] The maxillary index is obtained by dividing the length of the skull measured from the occiput to the premaxilla by the length of the superior maxilla. It indicates the amount of shortening which the maxilla has undergone relative to the whole cranium.

The Death Adder.

The forward rotation of the fangs in the death adder is produced by a combined forward and upward displacement of the maxilla and both of these are very obvious in the accompanying series of photographs.

In the resting position of the fangs it will be noted that the junction of the posterior arm of the maxilla and the ectopterygoid as indicated by the last of the post-orbital teeth, is situated about the middle of the inferior border of the orbit (see Figure XX), whereas in skulls showing elevation of the fangs it is located at the level of its anterior border (see Figures XXI and XXII).

Its upward displacement is equally obvious. Even at rest I have seen that the articulating surfaces of the prefrontal and the maxilla slope somewhat upwards and as the ectopterygoid impinges on the posterior arm of the maxilla, the latter slides forward and upwards over the surface of the prefrontal until it is checked by the ligaments of the prefronto-maxillary joint. This upward movement is rendered possible by bending at the ectopterygo-maxillary joint (see Figure XXI). As the forward pressure of the palatal arch increases, further bending at this joint takes place and other changes occur. The prefrontal, especially its inferior portion, is dragged forward and it is also displaced upwards. Widening of the orbit and an increase in the obliquity of its inferior surface which now looks more vertically forward, result (see Figure XXII). This permits further upward displacement of the maxilla, the inferior surface of which may now form an angle of approximately 50° with the horizontal. At rest the inferior surface of the maxilla is horizontal and the fangs point backwards at an angle of about 40°, but as a result of the above changes they present at an angle of about 90°, thus entering the bitten surface perpendicularly during the first stage of bite.

Weir Mitchell[3] described the points of the fangs in the rattlesnake as turning outwards during bite and regarded it as disadvantageous, inasmuch as it caused oblique entry and frequently threw one fang beyond the bitten part. In the skulls of some death adders and also tiger snakes prepared with the pterygo-palatine-transverse arch in a forward position, divergence of the fangs is often extremely well developed and this can also be observed in living snakes in whom the fangs during erection rotate outwards as well as forward. Where this occurs, oblique entry of the fangs must result in the first stage of biting. Its practical significance is considerable for much of the poison deposited in the tissues will lie outside the line of incision through the fang puncture. Excision under such circumstances is the rational mode of treatment.

Protraction of the pterygo-palatine-transverse arch which underlies the forward and upward dis-

placement of the maxilla, is associated with other changes shown in Figure XXII). These consist of a forward projection of the palatal bones on either side, the anterior extremities of which project almost as far forward as the anterior border of the premaxilla which is invariably displaced upwards.

The mechanism of elevation and forward rotation of the fangs in *Ancanthophis antarcticus* differs from that of the viper in which the movement of the maxilla on the prefrontal is essentially a rotatory one. In the death adder on the other hand it is chiefly produced by a forward and upward sliding movement of the superior surface of the maxilla on the inferior surface of the prefrontal, an accomplishment rendered possible by the bending of the posterior arm of the maxilla at the maxillary-ectopterygoid joint and the laxity of the prefrontal maxillary ligaments. Attention has been already directed to the fact that the articulating surface of the maxilla with the prefrontal is slightly concave in this species of colubride, so that a small degree of rotation probably also takes place. Its effect, however, is quite subsidiary to the chief mechanism as described above.

Its Relationship to the Solenoglyphæ.

The considerable degree of elevation of the fangs demonstrated in *Ancanthophis antarcticus* is only one of a number of characteristics linking it with the vipers and indeed by some of the older observers it was actually classified in that family. Its propinquity to the *Viperidæ* is shown in certain of its osteological features such as length of fang, shortening of the superior maxilla, scarcity of post-maxillary teeth (Table I), elongation of the quadrates (Table II) and flattening and broadening of the cranium. These features combined with its general physiognomy, the triangular shaped head, elliptical pupils, short tail and the rapidity of its strike, all of which are viperine characteristics, increase its resemblance to the *Solenoglyphæ* (see coloured plate). The grooved nature of its fangs and the anatomical configuration of the maxilla, however, necessitate its inclusion in the *Proteroglyphæ*. Biologically it is a snake of peculiar interest, for though it is not recorded beyond the confines of Australia and New Guinea, it probably approaches the vipers even more closely than either *Dendraspis angusticeps* and *Platurus colubrinus*. Its poison, however, is colubride in type and owing to its great toxicity, the large venom yield and the efficiency of the biting mechanism, the death adder is probably the most deadly of all terrestrial snakes.

The Other Poisonous Colubrides.

A mechanism very similar to that just described occurs in the case of the tiger snake, black snake and copper-head, except that the upward displacement of the maxilla is not as great nor the erection of the fangs as complete.

Notechis scutatus possesses the next most efficient elevating mechanism, but the fangs never become perpendicular, an angle of about 75° with the bitten surface being the maximum observed during the present series of observations, while the maximum angle of rotation does not exceed 30° to 35°.

The position of the fangs at rest and at maximal elevation is shown in Figures XVIII and XIX. Forward projection of the maxilla, as indicated by the relative position of the post-maxillary teeth, is well demonstrated in the two figures. Its increased obliquity and upward projection with rotation of the fangs are also evident as well as the usual upward displacement of the premaxilla associated with protraction of the palatine arch.

As is indicated in Table I the maxilla in the tiger snake is actually shorter than in any other colubrides studied, while the maxillary index shows that relative to the length of the whole skull its shortening is only surpassed by that of the death adder.

Skulls of the black snake (see Figure XVI) and copper-head were prepared with the pterygo-palatine-transverse arch at rest as well as in a forward position. Owing to the longer posterior arm of the maxilla, the post-maxillary teeth are never brought as far forward nor is the upward displacement as great as in the death adder. The fangs when fully rotated make an angle with the horizontal not exceeding 70°, the angle of rotation being approximately 25° to 30°. Photographs of the skull of a copper-head with reserve fangs and of a black snake with rotated fangs are reproduced in Figures XV and XVII).

In the brown snake rotation of the fangs is less than in any other Australian colubride and as if to compensate for this fact the fangs tend to be less recurved and the anterior end of the inferior surface of the maxilla is not quite horizontal in the resting position, but bends slightly upwards. The fangs are set at an angle of about 50° to the horizontal (see Figure XIV). Probably the angle of rotation through which the fangs pass does not exceed 15°.

From the data presented in Table I we see that a definite relationship exists between the maxillary index and the degree of elevation of the fangs. This is most developed in the death adder (3·7) which on this account and also because of the length of its fangs possesses an extremely efficient biting mechanism. The brown snake has an index of only 2·65. Its fangs are short, their elevation limited and its biting apparatus must be regarded as the least efficient of the colubrides under review.

The black snake and the copper-head have indices of 3·28 and 3·4 respectively and in regard to their capacity for forward rotation of the fangs, hold intermediate positions between the tiger and the brown snake. Owing, however, to the larger size of its fangs *Pseudechis porphyriacus* probably has the advantage as far as the actual inoculation of venom is concerned.

The Quadrate Index.

Physalix[4] states that the quadrate increases in length as one passes from the *Boïdæ*, through the

Proteroglyphæ to the *Solenoglyphæ*. In view of this transition it seemed important in these different Australian snakes to compare not only the absolute length of this bone, but the ratio of its length to that of the skull measured from the premaxilla to the occiput. The latter figure is called the quadrate index and the mean results of the various observations are incorporated in Table II. It will be seen that the quadrate index was 4·0 for the diamond snake, 3·8 for the carpet snake, 3·45 for the brown, 3·1 for the copper-head, 2·9 for the black snake, 2·5 for the tiger snake and only 2·0 in the case of the death adder. This means that relative to the length of the skull the quadrate proved half as long in the diamond snake as in the death adder, the other snakes occupying the positions indicated in Table II.

TABLE II.

The Relationship between the Length of the Quadrate Bone and that of the Skull in Certain Non-poisonous and Poisonous Australian Snakes.

Species of Snake.	Average Measurements in Centimetres.		The Quadrate Index (Average Results).
	Length of Skull.	Length of Quadrate.	
Diamond snake (*Python spilotes*)	4·5	1·12	4·0
Carpet snake (*Python spilotes* var. *variegatus*)	5·9	1·56	3·8
Brown snake (*Diemenia textilis*)	3·0	0·87	3·45
Copper head (*Denisonia superba*)	3·4	1·1	3·1
Black snake (*Pseudechis porphyriacus*)	4·1	1·4	2·9
Tiger snake (*Notechis scutatus*)	3·1	1·25	2·5
Death adder (*Ancanthophis antarcticus*)	3·5	1·75	2·0

The quadrate index = $\frac{\text{Length of Skull}}{\text{Length of Quadrate}}$. It indicates the shortening of the quadrate relative to the length of the whole cranium.

It is noteworthy that the quadrate and maxillary indices (Table I) are inversely related throughout the series, that is relative to the length of their crania the quadrate lengthens as the maxilla shortens in the different species. Here again a steady transition is noted in the Australian colubrides, the brown snake with its narrow head, long maxilla, numerous post-maxillary teeth and short quadrate approximating to the non-poisonous type, while the broad headed death adder approaches the *Viperidæ* which possess as a family the most perfect inoculation mechanism known.

The Structure of the Fangs.

The ophidia habitually kill and swallow large sized prey and inevitably teeth are lost in the process. In consequence large numbers of replacement teeth develope in reserve and the fangs which are specialized, grooved or canaliculated teeth, prove no exception to this rule. The presence of vertical tooth sacs and the mode of development by the familiar process of the formation of an enamel organ and dentine papilla has been worked out in detail by Tomes.[6]

Attention has been already drawn to the fact that two transversely arranged fangs are frequently found on the inferior surface of the one maxilla, but under such circumstances only one is functioning, the other being a reserve coming into position. In the Australian colubrides either the internal or the external socket may harbour the functioning fang, but Weir Mitchell[3] described the latter as invariably containing it in the rattlesnake. Bunched up and lying posteriorly are other successional fangs directed straight backwards, sometimes as many as six being observed in the death adder.

Grooving of the Fangs.

During development there appear on the lateral aspects of the anterior surface of the fang two ridges which elongate and become enfolded inwards until in the *Colubridæ* their rounded edges approximate except at the apex and base of the tooth. In the *Viperidæ* they actually fuse forming a canal, while in the *Hydrophinæ* and the *Opisthoglyphæ*, as Phisalix[4] has shown, the groove remains widely open throughout its whole length. Thus on section ophidian fangs show two cavities, an anterior groove or canal for conveying venom into the tissues and a more centrally placed pulp cavity containing blood vessels and nerves.

In the Australian colubrides on the anterior aspect of the base of the fang just below its point of ankylosis into the inferior surface of the maxilla is a V shaped opening through which the venom enters. The apex of the V is formed by the approximation of the two enfolded edges of the groove and for approximately two-thirds of its whole length this state of affairs persists. A horse hair passing through this groove is shown in Figure XXI. Functionally this groove acts as a canal and on penetration of the soft tissues external pressure by further approximating the edges ensures this result. In fangs embedded in paraffin the enfolded edges may actually overlap as a result of peripheral pressure.

At about its terminal fifth the groove reopens and becomes progressively shallower until near the apex it disappears. The accompanying series of photomicrographs selected from serial sections made through the grooved fang of a death adder illustrate these various changes (see Figures XXIV to XXX). Figure XXIV shows a section of the extreme base of the tooth at its attachment to the maxilla, while Figures XXV and XXVI are through the upper and lower V-shaped area described above. Figure XXVII shows the centre of the fang where the venom groove exceeds in its dimensions the pulp cavity. The last three sections (see Figures XXVIII, XXIX and XXX) show the gradual opening of the groove as the sharp apex of the fang is approached. These teeth are composed of dentine, pulp and a thin external layer of enamel and for purposes of comparison a section through the oval tooth of a non-poisonous carpet snake is shown (see Figure XXXI).

The grooved fangs of the other colubrides are similar in structure to that of the death adder and merit no special description.

Length of the Fangs.[1]

With the exception of the death adder all the common Australian colubrides are short fanged and this undoubtedly is an important factor in decreasing the efficiency of their bite.

In Table III the average length of the fangs measured by means of calipers from base to apex is detailed. The maximum and minimum lengths for different species are also included.

It will be seen that the average length of the fangs of the death adder was 6·2 millimetres (one-quarter of an inch), while the maximal and minimal lengths were 8·3 millimetres (one-third of an inch) and 5·0 millimetres (one-fifth of an inch) respectively. Next came the black snake with fangs whose average length was four millimetres (one-sixth of an inch), the maximum being five millimetres (one-fifth of an inch). The tiger and copper-head fangs averaged only 3·3 and 3·5 millimetres respectively, but in large sized specimens fangs of four to five millimetres are not uncommon. The brown snake on an average possessed the smallest fangs of the series, the average being only 2·8 millimetres (one-ninth of an inch), but in one large specimen they measured as much as four millimetres (one-sixth of an inch). It is interesting to note that this snake measured 160 centimetres (five feet three inches) in length and weighed 837 grammes, but nevertheless yielded no venom in captivity. The space separating its fangs was only 1·1 centimetres.

TABLE III.

Observations on the Length of the Fangs in Different Species of Australian Colubrides.

Species of Snake.	Number Examined.	Measurements in Millimetres.		
		Average.	Maximum.	Minimum.
Death adder ..	36	6·2	8·3	5·0
Black	11	4·0	5·0	3·5
Tiger	21	3·5	5·5	2·0
Copper-head ..	13	3·3	4·5	3·0
Brown	12	2·8	4·0	2·0

DENTAL IMPRESSIONS OF THE BITE.

Early in the course of the investigation it appeared to the writer that more accurate information might be obtained regarding dentition and the biting mechanism of the ophidia if a series of impressions of the teeth and buccal cavity could be taken in dental wax or some similar matrix.

Technique Employed.

After preliminary trial with the different commercial preparations commonly used in dental practice, Kerr's impression compound was selected as most suitable for the purposes of this inquiry.

In taking the bite a flat slab of this material four to eight millimetres in thickness was heated over a spirit lamp or in hot water until it had softened to a suitable consistency. The snake was first milked, then its jaws were drawn apart and the softened impression compound inserted into the buccal cavity. A snap bite often resulted as soon as the jaws were released and the mouth was now held firmly closed, while cold water was poured over the head to accelerate the consolidation of the impression and when this had ensued, it was carefully removed, first being displaced slightly backwards in order to insure clearance of the recurved teeth.

During the investigation a large number of these impressions was successfully taken and a series of photographs of actual bites in seven different species of Australian snakes is shown in Figures XXXIV to XLII. Eight of these show the impressions of the various teeth located in the upper jaw and roof of the mouth, but Figure XXXIX indicates the markings caused by the mandibular teeth of a death adder on the opposite side of the mould. The latter naturally are of minor import from the standpoint of the present investigation.

The Appearance of Dental Impressions.

The impressions reproduced in Figures XXXIV and XXXV are derived from non-poisonous snakes and indicate the typical U-shaped line of outer marginal teeth caused by the central pre-maxillary and the lateral maxillary punctures which number fourteen and sixteen in the carpet and eight and ten in the diamond snake for these particular specimens. More centrally situated are the two inner rows of markings due to the pterygo-palatine teeth.

Next comes the brown snake (see Figure XXXVI) with two fang marks on the right (one reserve) and one on the left side. Separated from these by about three millimetres are those produced by the post-maxillary teeth which number five and four on each side. The two inner rows of pterygo-palatine teeth, fifteen on the right and fourteen on the left side, are very evident. The impress of the lip margin can also be seen. It is interesting to note that in the act of biting some of the palatine teeth are inserted in front of the fangs.

The copper-head bite (see Figure XXXVII) shows the usual two prominent fang marks anteriorly situated, with four post-maxillary teeth marks on the right and three on the left as well as two long inner rows caused by the pterygo-palatine teeth.

The impression reproduced in Figure XXXVIII is from a black snake (*Pseudechis porphyriacus*). It presents punctures from three right and two left post-maxillary teeth with the two usual inner pterygo-maxillary rows.

The impression of a tiger snake bite is shown in Figure XL, in which the contour of the roof of the mouth, the anterior fang marks, the three post-maxillary teeth and the inner pterygo-palatines are clearly evident.

Figures XLI and XLII are derived from death adder bites. In each the impress and the lip margin and pterygo-palatine teeth are seen. One post-maxillary tooth mark is evident on each side

[1] A study of dental impressions of the bite and of amalgam and plaster of Paris models suggests that the whole length of the fang from apex to base is not available for penetration of the soft tissues. The *vagina dentis* even when rolled back still enfolds the basal fifth of the fang at its root and despite its compression against the bony maxilla must limit somewhat the depth to which the fang penetrates.

and the usual two fang marks are present in one (see Figure XLI), but not in the other (see Figure XLII) in which the left is missing. Here an indentation is seen unassociated with any puncture mark, a result due to the absence of a functioning fang on that side.

The Number of Fang Marks.

Tidswell[7] in his valuable clinical analysis of Australian snake bites eliminated from his series all cases which did not present the classical double puncture wounds in the bitten area, but this technique shows very clearly that the local lesion may present either one (see Figure XLI) or three fang punctures (see Figure XXXVI) caused by the bite of any poisonous snake. Even four punctures are possible from the one bite provided both reserve fangs are in position and sufficiently elevated, but this is a rare occurrence. Though the post-maxillary and pterygo-palatine teeth are very evident in dental impressions of the bite, they do not cause obvious skin lesions in man, the only teeth likely to do so, other than the fangs, being the anterior mandibulars of the death adder which are long, strong and prominent (see Figure XXXIX).

In the non-poisonous snakes on the other hand the U-shaped outline of the outer maxillary teeth is evident on the bitten skin and in large snakes like the carpet pterygo-palatine punctures may also occur. The buccal cavity of the ophidia is swarming with microorganisms and owing to the depth and number of the tooth punctures septic infection from bites of non-poisonous snakes is by no means unknown. Occasionally deaths have been reported from this cause.

The Distance Between the Fangs.

Stejneger[8] made the general statement that the distance between the two punctures would usually give a clue to the size of the snake and consequently to the presumable degree of poisoning, but no data were given to support this opinion nor was the relationship of interfang measurements to the species of snake or to the venom yield considered.

The introduction of the present technique has, however, enabled this to be done. In Table IV it will be noted that the average measurement separating the fangs during natural bite proved greatest in the death adder (1·6 centimetres) and least in the brown snake (0·9 centimetre), the dimensions for the tiger, black and copper-head being 1·4, 1·2 and 1·1 centimetres respectively. In a concurrent publication the correlation between the distance separating the fangs and the venom yield for snakes of the same species is clearly established, while it is also shown that in the different species of Australian colubrines under review the average venom yield increases *pari passu* with widening of the fang. Thus the broad headed death adder, though the smallest of the Australian venomous snakes, is the largest venom producer and the narrow headed large brown snake the smallest. The others occupy intermediate positions in this regard.

Again for any given species of snake the length of the fangs tends to increase as the distance between the fangs widens and as a direct consequence it follows that the distance between the skin punctures affords valuable clinical information regarding the dose of venom likely to have been inoculated.

The average number of teeth markings on the right and left sides of the mouth closely approximated, while in both the upper and lower jaws they were least numerous in the death adder and greatest with the brown snake and copper-head. In the tiger and black snakes intermediate results were recorded.

The Post-maxillary, Pterygo-palatine and Mandibular Teeth Marks.

Dental impressions, provided they are properly taken, afford an accurate index to the number of functional teeth in the buccal cavity, but undeveloped and very obliquely placed reserve teeth may not be recognized in the moulds.

In Table IV the bites in seventy-six Australian snakes of different species have been analysed. It will be noticed that all species show punctures due to the post-maxillary teeth, the average number increasing from the death adder (1·0-1·1) to the brown snake (5·5-6·2). For each species the average distance between the fang and the first post-maxillary tooth equalled three to four millimetres, the range of variation noted being from two to five millimetres.

The average number of the pterygo-palatine teeth as indicated in the moulds varied from 9·3 to 9·4 in the case of the death adder to 15·6 to 16·5 for the brown snake, while similar findings were recorded

TABLE IV.

Analysis of the Dental Impressions of the Bite of Seventy-six Australian Colubrides. (Functional Teeth in the Buccal Cavity.)

Species of Snake.	Number of Specimens Examined.	Average Distance in Centimetres Separating the Fangs.	Average Distance in Centimetres between the Fangs and the Post-maxillary Teeth.	Average Number of Post-maxillary Teeth.		Average Number of Mandibular Teeth.		Average Number of Pterygo-palatine Teeth.	
				Right.	Left.	Right.	Left.	Right.	Left.
Death adder ..	20	1·6	0·34	1·0	1·1	9·4	9·3	10·5	10·4
Tiger snake ..	20	1·4	0·37	2·5	2·6	12·8	13·1	13·1	12·9
Black snake ..	10	1·2	0·4	2·7	2·5	11·1	12·5	13·1	13·1
Copper-head ..	20	1·1	0·34	3·4	3·1	12·5	13·7	14·4	14·7
Brown snake ..	6	0·9	0·3	5·5	6·2	15·6	16·5	16·0	16·0

for the mandibular teeth being 9·3 to 9·4 in the former and 16·0 in the latter. The average number in the tiger snake, black snake and copper-head lay between 11·1 and 13·7 for the mandibular teeth and between 12·9 and 14·7 for the pterygo-palatines.

Reconstruction of the Position of the Fangs in Amalgam and Plaster of Paris Casts.

In this section of the work I am indebted to Dr. Mason Cox, D.D.Sc., for his invaluable advice and technical assistance in the construction of the amalgam and plaster of Paris models described

Figure XXXII.
A plaster-cast of the head and mouth parts of a death adder, showing the position of the fangs as in the first stage of biting.

below.[1] These models in the first instance were made from the dental impression of different species of snakes taken in Kerr's impression compound.

Two primary difficulties were encountered with amalgam casts, the first being due to air locks in the deep fang punctures preventing the amalgam from reaching the apices, while the second was due to fracture across the bases of the reconstructed fangs during the process of their separation in boiling water from the impression compound. These difficulties were largely overcome by placing one or two strands of fine copper wire in the fang punctures and adopting the procedure of tamping the amalgam which brought it to the extreme limits of the apical impressions in a manner that packing entirely failed to do. Mr. W. Stone also demonstrated the superiority of this technique in fine capillary glass tubes which could not be filled with amalgam by any process of mechanical or instrumental packing, but which was readily accomplished by tamping.

In models reconstructed in this manner the post-maxillary and pterygo-palatine teeth as well as the fangs were clearly reproduced. The recurved fangs, however, generally pointed backwards at an angle exceeding that which direct observation had suggested would be the case. As a general rule especially with the death adder but also with the tiger, black and copper-head snakes the fangs appeared to enter the impression compound in an elevated position, yet the models gave no indication of this for the reconstructed fangs pointed backwards.

A series of impressions was then taken in recently killed snakes with the mouth partially opened and with the pterygo-palatine-transverse arch in both forward and backward positions. Water soluble commercial plaster was used for this purpose. After this had hardened it was left *in situ* and a complete external mould of the whole head was built up in Kerr's impression plaster in four sections to facilitate their removal intact.

In reconstructing the model the external mould was filled with artificial stone (Plaston) and the water-soluble mould of the buccal cavity was keyed in its correct position. After setting, the four sections of the impression plaster were removed and the water-soluble plaster was dissolved out, leaving a true model of the buccal cavity in artificial stone in correct relationship to the head.

In models of this nature it was clearly demonstrated that the position of the fangs varied with the degree of advancement of the pterygo-palatine-transverse arch.

Thus on its full protraction the fangs showed maximal elevation (see Figure XXXII) and on retraction they pointed obliquely backward (see Figure XXXIII). The latter corresponded to the condition found in many of the amalgam models reconstructed from Kerr's impression compound and the only explanation of this apparent anomaly was that at the completion of a natural bite the fangs pointed downwards and backwards owing to retraction of the pterygo-palatine-transverse arch.

The Musculature of the Australian Colubrides.

The death adder may be taken as a type for it undoubtedly possesses the best muscular development and the most efficient biting mechanism of any of the Australian colubrides. The following description is primarily based on the musculature of this snake, attention being directed only to the more notable features of divergence met with in the other species.

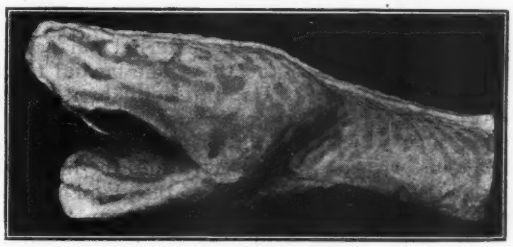

Figure XXXIII.
Showing maximal retraction of the fangs at the completion of the bite.

The action of different muscles has been ascertained experimentally by electrical stimulation in snakes after destruction of the brain and throughout this whole series of observations the invaluable work of Phisalix[4] on the musculature and mechanism of bite in colubrides in other parts of the world has been confirmed.

Muscles Opening the Mouth.

The muscles involved in depressing the lower jaws are chiefly the digastric (see Figure V at 20),

[1] A series of these amalgam and plaster of Paris models were demonstrated at the October meeting of the Victorian Branch of the British Medical Association.

the vertebro-mandibular and the costo-mandibular muscles. Noguchi[2] states that the function of the *digastricus* is to fix the articulation of the jaw, but the result of electrical stimulation in the Australian colubrides shows it to be a powerful elevator of the mandible as Phisalix[4] and others have long held. On contraction owing to its attachments to the posterior aspect of the squamosal on the one hand the upper border of the posterior terminal extremity of the mandible on the other, it must elevate the latter and in so doing depress the whole of the mandible situated anteriorly to the quadrato-mandibular joint. Also, as shown by Phisalix, it is the cervico-angular muscle of Duveroy and the sub-occipito-articular muscles of Dugès (see Figures IV and V at 21) which fix the lower end of the quadrate and so permit the mandible to rotate around its inferior condyle as a fixed point.

Muscles Closing the Mouth.

The group of muscles closing the mouth, that is elevating the mandibles, are the anterior, middle and posterior temporals and the first of these is specially important owing to its relationship to the venom gland.

In the *Boïdæ* the anterior temporal (mandibular of Owen) takes origin chiefly from the anterior aspects the parietal bone just behind the orbit and passes uninterruptedly downwards and slightly backwards to be inserted into the outer surface of the ramus of the mandible behind the mandibular teeth. In the colubrines the presence of the venom gland has led to the division of this muscle into a superior portion inserted into the gland capsule and an inferior portion taking origin from the same structure.

The former arises from the external surface of the post-orbital process and the whole medial ridge of the parietal bone. Its anterior fibres sweep downwards and backwards, its middle fibres directly downwards and its posterior fibres downwards and forwards to be inserted into the internal, superior and posterior surfaces of the gland capsule (see Figure I at 1). On electrical stimulation of these fibres the gland is twisted and compressed as well as being drawn sharply upwards and somewhat inwards.

The inferior portion of this muscle has a limited tendinous origin from the posterior third of the internal surface of the gland capsule. It then sweeps downwards and forwards round the lip margin to be inserted into the outer border of the middle third of the mandible (see Figure I at 2). When these fibres are stimulated the posterior portion of the gland is compressed and twisted downwards. The simultaneous action of the superior and inferior divisions of the anterior temporal muscle results in the instantaneous ejection of a considerable proportion of the gland contents, especially as the external pterygoid muscle (see Figure I at 4), on which the gland partially lies, also assists in its compression.

The middle temporal muscle is deeply situated, being completely covered by the anterior temporal both at its origin and for most of its course. It has an extensive fan-like origin from the superior surface of the parietal bone external to the origin of the anterior temporal muscle, extending from in front of an external parietal process of bone[1] anteriorly to the squamosal bone posteriorly. Passing downwards and slightly backwards it narrows somewhat and is inserted into the superior border of the mandible just posterior to the anterior temporal muscle.

The posterior temporal has a limited origin mainly from the outer surface of the squamosal bone and the quadrate. Its curved anterior border lies in contact with the anterior temporal muscle and its posterior border with the latter bone. It is inserted into the external surface of the posterior third of the mandible, its anterior half completely covering the middle temporal muscle in this situation (see Figure I at 3).

In the brown snake the anterior temporal muscle is less well developed, its fibres of origin not extending as far back on the parietal bone as in the other four species of Australian colubrides. In consequence its upper fibres are not inserted into the posterior surface of the gland capsule and it only very incompletely covers the middle temporal muscle, an extensive triangular shaped area of which is visible on reflecting the skin. In this respect we again perceive the approximation of the brown snake to the non-poisonous *Aglyphæ* and *Boïdæ* in which the middle temporal muscle remains uncovered.

The Protractors of the Pterygo-palatine-transverse Arch.

Two most important muscles in the dynamics of the bite are the spheno-pterygoid and the parieto-pterygoid, for they alone on electrical stimulation cause forward movement of the palatal arch with elevation of the fangs.

The spheno-pterygoid arises from the basi-sphenoid and passes backwards, downwards and outwards to be inserted into the internal and upper aspect of the posterior portion of the endopterygoid.

Its relationships may be studied in the accompanying drawings prepared from dissections of the death adder (see Figure IV) and the tiger snake (see Figure V).

The parieto-pterygoid muscle originates from the parietal bone somewhat anterior to and at a deeper level than the spheno-pterygoid does from the basi-sphenoid (see Figure V at 19). It spreads outwards, downwards and backwards in fan-like fashion to be inserted into the inner and upper aspect of the middle third of the endopterygoid.

[1] An external parietal process is a characteristic osteological feature of the death adder and is only slightly in evidence in the other colubride skulls. It is an external projection of bone situated some four millimetres behind the post-orbital process. The fibres of the middle temporal muscle sweep round behind it in their course to the mandible.

The Retractors of the Pterygo-palatine-transverse Arch.

The chief retractor muscles are the parieto- or spheno-palatine and the internal and the external pterygoids. The former muscle acts on the palatine bone, the latter two on the pterygoid and the maxilla.

The Parieto-palatine.

The parieto-palatine muscle arises in the death adder from the parietal bone adjacent to, but at a deeper level than the basi-sphenoid (see Figures IV and V at 13). At its origin it is covered by the spheno-pterygoid muscle whose fibres cross approximately at right angles. The parieto-palatine muscle takes an outward, downward and forward course to be inserted into the internal and superior surface of the palatine bone just anterior to the endopterygo-palatine junction, while a superior strand of muscular fibres passes on over this bone to the inner aspect of the superior maxilla. The fibres from this muscle run in the *vagina dentis* and act as tensors of the edges of the sheath during the act of biting.

The External Pterygoid.

The external pterygoid muscle arises from the external and inferior surface of the terminal posterior extremity of the mandible close to its junction with the quadrate and sweeping under the joint forms a massive muscular swelling on the lateral aspect of the palate (see Figures IV and V at 4). It lies external to the pterygoid bone with the internal pterygoid muscle on its inner and the mandible and posterior and middle temporal muscles on its outer side and proceeds directly forwards to be inserted into the external process of the ectopterygoid (see Figure II at 4). The latter is only met with in *Ancanthophis antarcticus* and is a smooth protuberance of bone which projects outwards and backwards from the ectopterygoid about six millimetres posterior to the ectopterygo-maxillary joint. It is not met with in the skulls of the other Australian colubrines and in these the external pterygoid has a tendinous insertion into the outer surface of the ectopterygoid in the vicinity of its junction with the posterior arm of the superior maxilla. Naturally the length of this muscle varies in different snakes according to the length of the latter bone. It is shorter in the case of the brown snake than the other colubrides, while in the non-poisonous carpet and diamond snakes its relative length is still more limited. In the *Viperidæ* on the other hand the external pterygoid is powerfully developed and is inserted far forward into the superior maxilla itself.

The inner aspect of the inferior surface of the venom gland partially rests on the external pterygoid (see Figure I at 4). This relationship is of considerable importance since the pressure which it exerts from below upwards assists in the expulsion of the gland contents during bite and naturally the better developed the muscle the more significant its rôle in different species of snakes.

The Internal Pterygoid.

The internal pterygoid muscle arises from the inferior surface and external border of the endopterygoid posterior to its junction with the transverse bar. It proceeds backwards and outwards converging to be inserted into the internal aspect of the terminal posterior extremity of the mandible opposite the origin of the external pterygoid.

Phisilix[4] pointed out that if the quadrato-mandibular articulation is immobilized by the contraction of the cervico-angular muscle the posterior extremity of the mandible becomes fixed. Both the internal and external pterygoids are attached to this bone and their contraction will consequently have the effect of dragging backward the entire pterygoid-palatine-transverse arch. If on the other hand the articulation is rendered mobile by relaxation of the cervico-angular muscle and if the pterygo-palatine transverse arch be immobilized by the implantation of teeth into its prey, then contraction of the pterygoid muscles will have the effect of drawing the mandibles forward, a movement which is of considerable importance in the act of swallowing any large sized object. This mechanism holds for the Australian colubrides and the two distinct actions dependent on the mobility or the immobility of the quadrato-mandibular joint can be demonstrated by experimental stimulation of the pterygoid muscles.

The Mechanism of Bite.

Like other snakes many of the Australian colubrides when annoyed emit a hissing sound due to the expulsion of air under pressure through the slit-like orifice of the trachea. The increased aeration may possibly subserve a useful purpose by placing them physiologically in a better position for either flight or fight.

Another feature is the cobra-like flattening of the neck which occurs when these snakes are disturbed and angry. This is most evident in the case of the black and tiger snakes and is produced by an elevation of the ribs. By electrical stimulation of the adjacent muscles I have reproduced this movement in snakes the brain of which had been destroyed. In all essentials it corresponds to the expansion of the hood observed in the cobra under analogous circumstances. A somewhat similar flattening occurs in the copper-head, but it is less localized, extending downwards for a greater distance (twenty-two to thirty centimetres), while in the death adder a flattening out of the whole body occurs. In the brown snake on the other hand flattening is less obvious and very limited, extending for a distance of only five or six centimetres behind the head.

The Strike.

When a poisonous snake strikes, it throws itself forward with considerable rapidity and violence, but the distance covered does not exceed one-third of its length. Of the Australian colubrides the death adder is by far the fastest striker, doing so with amazing rapidity though not attaining the colossal speed of the Russell's viper.

All these snakes strike with the mouth closed except the brown snake (*Diemenia textilis*) which does so with the jaws open. Other peculiarities of this snake are the S-like curve to the right which it makes before striking and also the degree of elevation of the head which is raised about thirty centimetres.[1] These peculiarities no doubt help to compensate for the very limited rotation of the short fangs during biting.

The tiger and black snakes raise the head some fifteen to twenty centimetres in striking, but the death adder and also the copper-head generally strike while flat on the ground.

Opening of the Mouth and Elevation of the Fangs.

As the head of the snake reaches its victim, the mandibles are depressed by the rapid contraction of the digastric, the cervico-mandibular and vertebro-mandibular muscles and simultaneously the fangs are rotated forward by the forward swing of the pterygo-palatine-transverse arch. This is brought about by the simultaneous contraction of the spheno-pterygoid and parieto-pterygoid muscles. The angle of rotation of the fangs varies with the species of snake as has already been described, being greatest in the death adder (40° to 50°), of moderate grade in the tiger, black and copper-head (25° to 35°), while in the brown snake it is minimal (10° to 15°).

Closure of the Mouth and the Injection of Venom.

The next stage is closure of the mouth which is brought about by the simultaneous contraction of the anterior, middle and posterior temporal muscles which strongly elevate the mandibles. These muscles are powerfully developed in most of the Australian colubrides and it may take a considerable degree of muscular effort to dislodge a death adder or copper-head once it has engaged its prey.

With closure of the jaws and the entry of the fangs immediate inoculation of venom occurs. This is brought about mainly by the contraction of the superior and inferior portions of the anterior temporal muscle which compress the gland by producing torsion on its capsule with the expulsion of venom from the acini into the duct. The venom passes through the dental papilla into the *vagina dentis* which, by the tense approximation of its edges to each other and to the surface of the fang, prevents escape of the poison except through the V-shaped opening on the anterior groove of the base of the latter. From here it is conveyed under pressure through the groove into the tissues. Martin[9] regarded the pulling forward of the anterior surface of the base of the fang against the poison papilla as assisting in this mechanism, the exactness of which was illustrated by the fact that in only two instances was leakage in this situation ever observed.

Acton and Knowles[10] stress the importance of fixation of the lower jaw if colubride bites are to be effective and owing to the anatomical insertion of the anterior temporal into this bone this undoubtedly is an important factor in the injection of a full dose of venom. With the Australian colubrides, however, venom may sometimes spurt out for a considerable distance even where no actual object is bitten, a result which is probably attributable to the vigorous contraction of the temporal muscle on a full gland. Electrical stimulation of the upper portion of the anterior temporal muscle even in the absence of contraction of its lower fibres produces much compression and torsion of the gland of a magnitude sufficient to cause definite expulsion of venom.

Retraction of the Fangs.

Immediately following the entry of the rotated and elevated fangs and actually accompanying the discharge of venom there occurs another set of movements due to contraction of the retractor muscles operating on the pterygo-palatine-transverse arch which results in the distribution of venom along an oblique posteriorly directed fang track. When the quadrato-mandibular joint is fixed, contraction of the parieto-palatine and the internal and external pterygoid muscle leads to retraction of the arch and with it the fangs which, though they enter the tissues in a position of maximal elevation, continue their subsequent course in a downward and backward direction. Evidence of this fact is shown in models reconstructed with the palatal arch in a forward position on the one hand and in natural bites on the other (see Figures XXXII and XXXIII). The extreme degree of retraction which the fangs may undergo in the death adder, is here depicted. The degree of posterior obliquity of the fangs is greater than in the resting position and as in the vipers overaction is prevented by the anterior prefronto-maxillary ligaments. Small animals may actually be observed to be drawn further backwards into the mouth during this movement, while in large sized animals it has the reverse effect of drawing the head of the snake forward instead. Two other functions of the retractor muscles also come into play during this movement. The first is due to the contraction of the superior bundle of the parieto-palatine muscle which passes outwards over the palatine bone on to the internal surface of the superior maxilla supplying fibres acting as tensors of the *vagina dentis*. The other is the compressor action exerted by the contraction of the external pterygoid on the inferior surface of the venom gland.

This action, synchronizing as it does with the contraction of the anterior temporal muscle, facilitates the injection of venom under pressure.

Though the four stages including the inoculation of venom have been described separately, in nature they occur as a series of rapidly coordinated movements, many lethal doses being injected in a fraction of a second in some instances.

Withdrawal of the Head.

Relaxation and disengagement of the head occur at a variable period after the whole mechanism of

[1] My attention was first drawn to these interesting facts by Mr. T. Eades who possesses a wide knowledge of the habits of the Australian ophidia.

bite has been completed. It is accomplished by a reversal of the above processes, the palatal arch being protracted and the fangs elevated, the mandibles depressed and the head finally withdrawn.

The Voluntary Control Exerted by the Snake During Biting.

The degree of control exerted by the colubrides over the venom gland and the inoculation mechanism during biting is considerable. The forward rotation of the fangs is under voluntary control and is independent of the act of opening the jaws. Furthermore, even after penetration with the fangs erect, the snake can accurately control the amount of venom injected, this being largely determined by the intensity of the muscular contraction of the anterior temporals.

Unilateral control is complete and the contents of only one of the venom glands may be injected at a natural bite, the other being held in reserve. It is thus evident that even where fixation of the lower jaws has been accomplished, only a proportion of the total venom may be injected. On the other hand, these colubrides often hang on to the bitten object with great tenacity and one may observe several independent contractions of the temporal muscles occurring either synchronously or alternately with the object of injecting the whole of the contents of the venom glands.

Thus, apart from the length and elevation of the fangs, the fixation of the lower jaw and the amount of venom present in the glands, the voluntary power exerted by each individual snake comes into play during natural bite.

Summary and Conclusions.

1. A new method of studying ophidian dentition and the mechanism of bite is described.
2. Quadrate and maxillary indices are introduced for purposes of comparing certain osteological features of the skulls of Australian snakes.
3. The significance of interfang measurements is discussed.
4. The function of the various muscles operating on the venom gland and the pterygo-palatine-transverse arch was confirmed by electrical stimulation in specimens after destruction of the brain.
5. In order of biting efficiency the death adder is first, the brown snake last and the tiger snake, black snake and copper-head occupy intermediate positions.
6. Data are presented showing that the brown snake links up with the non-poisonous colubrides (*Aglyphæ*) and the death adder with the vipers. The last snake possesses a considerable degree of forward rotation of the fang.

Acknowledgements.

I am indebted to Miss Cecil Maudsley for the excellent series of photographs and photomicrographs reproduced in the article. The drawings on ophidian musculature were made by Mr. A. T. Mockridge from freshly dissected preparations made at the institute.

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British Medical Association News.

MEDICO-POLITICAL.

A meeting of the Victorian Branch of the British Medical Association and of the Medical Society of Victoria was held at the Medical Society Hall, Albert Street, East Melbourne, on February 6, 1929, Mr. B. T. Zwar, the President, in the chair.

Presentation of Portrait of the Late Charles Ryan.

A portrait of the late Charles Ryan was presented to the Branch. In making the presentation on behalf of the subscribers Dr. Felix Meyer said that the name of Charles Ryan recalled to those of the older generation a very real and living personality.

Charles Ryan was a surgeon of distinction. His surgery reflected the character of the man in its courage, resourcefulness, and decision. He was a great nature lover, fond of sport and of the open air, full of the joy of life and enthusiasm for all its activities. His genuine sympathy and charm of manner completed the picturesque personality of a man richly endowed with gifts of head and heart, of whom the Athenians would have said that he was beloved of the Gods because he died young at heart.

Sir George Syme said it was his esteemed privilege to be associated with the presentation. Dr. Felix Meyer had touched a note of high seriousness which fitted the occasion. Charles Ryan had occupied a high position in the community. In all his work he had been actuated by a very strong sense of duty. Particularly did this apply to his military service. He had served as an honorary surgeon to the Melbourne Hospital and the Children's Hospital long and faithfully and had there done some of his best work.

He had also laboured well in the service of the British Medical Association and the Medical Society of Victoria, had held high office and had contributed many scientific papers.

In all things he had maintained the highest traditions of the profession. Sir George Syme joined with Dr. Meyer, Dr. Fetherston and many other old friends in asking the President on behalf of the Branch to accept the portrait of a man of high attainments and unique personality, a very eminent member of the profession.

VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

STATEMENT OF RECEIPTS AND PAYMENTS FOR PERIOD FROM JANUARY 11, 1928, TO JANUARY 9, 1929.

RECEIPTS.	£ s. d.	£ s. d.
Jan. 11, 1928.		
To Balance, National Bank of Australasia Ltd.	90 16 6	
„ Cash in Hand	12 0 0	
		102 16 6
Jan. 9, 1929.		
To Subscriptions—		
Metropolitan Members	3,482 19 3	
Country Members	1,690 5 3	
Junior Members	351 5 0	
Capitation Grant, London *et cetera*	3 4 6	
		5,527 14 0
„ Clerical work for Members	74 12 7	
„ Clerical, Obstetric Research Committee	83 6 8	
„ Clerical, Permanent Post-graduate Committee	25 0 0	
		182 18 3
„ Refund Expenses *re* Hall		19 10 0
„ Sale, Hospital Report Forms		0 9 0
		£5,833 7 9

PAYMENTS.	£ s. d.	£ s. d.
Jan. 9, 1929.		
By Medical Society of Victoria		2,707 16 9
„ *The British Medical Journal*		1,702 0 3
„ Salaries—		
Assistant Secretary	240 10 0	
Assistant Librarian	169 0 0	
Junior Clerk	65 4 0	
		474 14 0
„ Rebates to Divisions		92 1 9
„ Postages, Duty Stamps, *et cetera*		71 17 11
„ Printing		62 6 6
„ Stationery		67 17 11
„ Commission on New Members		5 5 0
„ Advertising		9 8 0
„ Audit Fee, 1927		5 5 0
„ Telephone		9 10 8
„ Clerical Assistance		4 0 6
„ Electric Light and Power		15 19 8
„ Legal Expenses		3 3 0
„ Repairs *et cetera*		17 13 11
„ Travelling Expenses		0 16 2
„ Bank Charges		14 18 4
„ Insurance		1 3 4
„ Freight on Journals	2 14 2	
„ Expenses *re* Yallourn Mtg.	5 10 0	
„ Expenses *re* Prof. Wollard Mtg.	10 6 0	
„ Expenses *re* University Mtg.	5 2 0	
„ Expenses Federal Committee Representative	12 18 0	
„ Lanternist *et cetera*, Branch Mtg.	4 1 5	
„ Hire of Chairs	4 5 0	
„ Acts, Regulations and Newspapers	2 3 0	
„ Sundries	3 11 2	
		50 10 9
„ Balance, National Bank of Australasia Limited	504 18 4	
„ Cash in Hand	12 0 0	
		516 18 4
		£5,833 7 9

Compared with the Books and Accounts of the Victorian Branch of the British Medical Association and found to be in accordance therewith.

J. V. M. WOOD & Co., F.I.C.A.,
Incorporated Accountants and Auditors.

C. STANTON CROUCH,
Secretary.
CRAWFORD H. MOLLISON,
Hon. Treasurer.

Melbourne, February 4, 1929.

MR. B. T. ZWAR, the President, accepted the portrait on behalf of the Branch.

Financial Statements.

The Honorary Treasurer, DR. C. H. MOLLISON, presented the balance sheets of the Victorian Branch of the British Medical Association and of the Medical Society of Victoria. The balance sheets were adopted.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Rutherford, Lawrence Osborne, M.B., 1927 (Univ. Sydney), Wentworth Street, Point Piper.

Malcolm, Robert James Wherry, M.B., Ch.M., 1924 (Univ. Sydney), Coast Hospital, Little Bay.

The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Sandner, Eugene, M.B., B.S., 1928 (Univ. Melbourne), Bendigo.

Thorburn, Ian Oriel, M.B., B.S., 1928 (Univ. Melbourne), Melbourne Hospital.

Farran, Egbert Armytage Cunninghame, M.B., B.S., 1928 (Univ. Melbourne), Melbourne Hospital.

Robinson, Norman Henry, M.B., B.S., 1928 (Univ. Melbourne), Melbourne Hospital.

Brotchie, Edward Bonaventure Heffernan, M.B., B.S., 1928 (Univ. Melbourne), Fitzroy.

Allen, Thomas Gilmour Bowen, M.B., B.S., 1928 (Univ. Melbourne), Melbourne Hospital.

Amies, Arthur Barton Pilgrim, L.R.C.P. *et* S. (Edinburgh), L.R.F.P.S. (Glasgow), 1928, 32, Collins Street, Melbourne.

MEDICAL SOCIETY OF VICTORIA.

STATEMENT OF RECEIPTS AND PAYMENTS FOR PERIOD FROM JANUARY 11, 1928, TO JANUARY 9, 1929.

RECEIPTS.	£	s.	d.
Jan. 9, 1929.			
To Subscriptions	2,707	16	9
„ Transfer from Medical Agency on account of Building Fund	417	17	6
	£3,125	14	3

PAYMENTS.	£	s.	d.	£	s.	d.
Jan. 11, 1928.						
By Overdraft at National Bank of Australasia				1	2	10
Jan. 9, 1929.						
By THE MEDICAL JOURNAL OF AUSTRALIA				1,636	10	0
„ Interest on Building Fund Debentures				442	15	0
„ Redemption of Building Fund Debentures				200	0	0
„ Salaries—						
Secretary	412	0	0			
Library Clerk	60	0	0			
Caretaker	100	0	0			
Bonuses to Staff	22	10	0			
				594	10	0
„ Library, Journals and Binding				50	2	10
„ Furniture				9	2	6
„ Postages				71	17	10
„ Rates, Taxes and Insurance				43	3	1
„ Light and Power				15	19	8
„ Audit Fee, 1927				5	5	0
„ Bank Charges				1	0	0
„ Telephone				9	10	8
„ Repairs—						
Epidiascope	3	6	6			
Electrical	8	6	3			
Plumbing	2	8	0			
Furnishings	1	2	6			
				15	3	3
„ Sundries				4	6	6
„ Balance				25	5	1
				£3,125	14	3

BALANCE SHEET AT JANUARY 9, 1929.

LIABILITIES.	£	s.	d.
Debentures, Building	6,125	0	0
Balance	4,428	8	6
	£10,553	8	6

ASSETS.	£	s.	d.
Balance, National Bank of Australasia Ltd.	203	6	3
Building	9,203	6	3
Furnishings and Fittings	1,324	17	2
	£10,553	8	6

Compared with the Books and Accounts of the Medical Society of Victoria and found to be in accordance therewith.

J. V. M. WOOD & Co., F.I.C.A.,
Incorporated Accountants and Auditors.

C. STANTON CROUCH,
Secretary.

CRAWFORD H. MOLLISON,
Hon. Treasurer.

Melbourne, February 4, 1929.

Medical Societies.

THE MELBOURNE PÆDIATRIC SOCIETY.

A MEETING OF THE MELBOURNE PÆDIATRIC SOCIETY was held at the Children's Hospital, Carlton, on June 13, 1928.

Spinal Deformity.

MR. H. DOUGLAS STEPHENS showed a boy, A.B., aged seven years, with spinal curvature. Two months previously he had been feverish for a few days and had developed tonsillitis. Four weeks later it was noticed that he had developed some curvature of his spine and since then this abnormality had become more evident. As a matter of interest it was stated that six months before a child living next door to this boy had had infantile paralysis. Mr. Stephens doubted if the spinal abnormality in his patient had developed in the two months. He thought that probably it had been present for some time before its recognition.

DR. H. B. GRAHAM thought that the condition was probably an old poliomyelitis which had not been recognized at the time.

MR. RUPERT DOWNES, D.S.O., thought that the condition was not one of poliomyelitis, but was simply a static scoliosis.

DR. LIONEL HOOD pointed out that it was very rare for a patient with poliomyelitis to have only the dorsal muscles affected.

MR. STEPHENS in discussing treatment suggested that the child be put on a Lovett's frame for six weeks and after that time a commencement be made with massage and exercise.

Infantile Scurvy.

Mr. Stephens also showed a second patient, K.P., a male baby, aged ten months, who had advanced bony changes probably due to scurvy.

He had been admitted to hospital on April 21, 1928, on account of a painful swelling just above the left knee. This condition had been present for three weeks and had very slowly increased in size during that time.

On admission the diagnosis lay between scurvy and osteomyelitis and an operation had been undertaken in order to clear up the doubt. Blood clot was found which had stripped up the periosteum over the lower third of the left femur, but there was no evidence of any suppurative process and the wound had been closed. The infant had been given large doses of orange juice and cod-liver oil emulsion and although the temperature frequently reached 38·3° C. (101° F.) during the first ten days, the general condition had improved gradually. In spite of this improvement, however, during the first few days in hospital a swelling had been noticed over the lower third of the right femur and this became more pronounced. After a further three weeks bilateral swellings had been noticed over the heads of each humerus. X ray examination revealed a raising of the periosteum with new bone formation at each of the areas where a bony swelling was present. There had been no reaction to the Wassermann test of the infant's blood.

DR. H. LAWRENCE STOKES expressed the opinion that the condition was one of scurvy and mentioned that he was surprised that further scorbutic lesions should have manifested themselves during the period that orange juice had been given. In support of his opinion he pointed out that the X ray films showed definite decalcification of all the epiphyses, the centres being pale and homogeneous and each surrounded by a thin layer of compact bone. At the same time there were also definite rhachitic changes.

DR. J. W. GRIEVE supported Dr. Stokes.

MR. C. J. O. BROWN thought that the epiphysis at the lower end of the right femur was separated from the shaft because the periosteum was separated below the epiphyseal line.

DR. LIONEL HOOD thought that the condition was specific and he pointed out that the child had a facies that suggested congenital syphilis.

Ankylosis of the Temporo-mandibular Joint.

MR. RUPERT DOWNES showed a male child, D.L., aged five years, with an ankylosis of the left temporo-mandibular joint. Two months previously the patient had *otitis media* which had been followed by meningitis. A swelling in the region of the temporo-mandibular joint on the left side had appeared with limitation of movement of the joint. Dense bone formation only was seen in the skiagram in the region of the joint. Mr. Downes thought that the condition was probably an arthritis, pyogenic in nature. He intended to examine the child under an anæsthetic in order to determine the full range of movement. With regard to treatment he thought that he would first try the effect of graduated wedges inserted between the teeth. If these were unsuccessful, he intended to perform an arthroplasty and finally to excise the condyle if necessary.

MR. H. D. STEPHENS mentioned two somewhat similar cases he had treated. In one he had used wedges and in the other he had gouged out the joint, but the trouble returned. He considered that arthroplasty was the best treatment.

Renal Neoplasm.

DR. J. W. GRIEVE showed a female child, E.T., aged two years and three months, with a tumour in the left upper segment of the abdomen. The development and health of the patient during the period of infancy had been normal. A few months previously a swelling had appeared in the left hypochondrium. The tumour was painless and had steadily increased in size. Dr. Grieve thought that the condition was probably a renal sarcoma.

MR. R. M. DOWNES also thought that the condition was malignant and was probably a Wilm's embryonic tumour. He suggested that X rays should be given therapeutically.

DR. LIONEL HOOD supported the two previous speakers with regard to the neoplastic nature of the tumour. He thought that a pyelogram should be undertaken and also a urea concentration test.

MR. C. J. O. BROWN pointed out that a Wilm's tumour would react within a few weeks to X ray exposures. If these were carried out, the result might possibly help in clearing up the diagnosis.

MR. H. D. STEPHENS agreed with Dr. Grieve's diagnosis. He also thought that there were already secondary growths in the retroperitoneal glands and needless to say the prognosis was very bad. He had removed a kidney when there was not a trace of any secondary growth and yet within ten months the whole abdomen was studded with sarcomatous growths. Most of the patients he had seen had been under the age of three years. He suggested the implantation of radium needles.

Dr. Grieve quoted the case of a patient he had seen some years before at the Children's Hospital. She was a girl, aged nine years, with a renal sarcoma, which condition had been greatly reduced following X ray exposures and for some months she was much improved.

Congenital Syphilis.

Dr. Grieve's second patient was a baby only a few weeks old. Snuffles had been present from birth and the infant's skin was very papery. The serum of both the baby and the mother had yielded Wassermann reactions. Dr. Grieve showed this patient because it had an enormous spleen extending down to the *symphysis pubis*; he wondered if the syphilitic condition itself could account for this enlargement.

DR. B. L. STANTON suggested that a splenic puncture should be performed for diagnostic purposes.

DR. W. MCLAREN asked why more syphilitic stigmata were not present, but Dr. Grieve pointed out that the child had a shiny, scaly skin.

MR. STEPHENS asked if such a child was liable to develope Banti's disease.

DR. M. TALLENT quoted two cases of congenital syphilis which had been recently treated at the hospital with "Sulfarsenol"; each patient became worse and died, probably from œdema around the anterior horn cells.

Pathological Specimens.

DR. REGINALD WEBSTER then showed some very interesting pathological specimens.

The first was a larynx from a child, aged four years, who had died from laryngeal diphtheria which was contracted shortly after an operation for repair of a cleft palate.

The second was a left kidney which had been removed from a child, aged six years, who since infancy had had recurring pain in the left side and who frequently passed pus in his urine. The specimen showed a unilateral hydronephrosis which was probably caused by a valve at the upper end of the ureter.

The third was a heart of a child, aged eleven years, in which there was a large deficiency in the interventricular septum. Dr. Webster asked if any member had seen a child with such a heart who had lived longer than eleven years.

The fourth was also a heart, a typical example of subacute bacterial endocarditis. There were very definite profuse vegetations on the mitral valves. The spleen was also shown with typical infarcts. There was no history suggesting that the child had ever had rheumatic fever.

Dr. Webster mentioned the case of a child he had seen which was supposed to be suffering from subacute bacterial endocarditis; non-hæmolytic streptococci had been found on blood culture. *Post mortem* examination, however, had revealed that the heart was normal and that the child had died from broncho-pneumonia.

The fifth was still another heart from a child, aged thirteen years, affected with mitral stenosis and aortic regurgitation. There was a definite history of repeated rheumatic infections during the previous three years.

The sixth was the brain of a child who had died from a cavernous thrombosis following an injury which became infected with *Staphylococcus aureus*. There were infected infarcts in the left lung and in the kidneys.

Mr. Downes said he had recently seen a patient who had developed a cavernous sinus thrombosis with complete blindness and proptosis and who recovered. He said that there were a number of such cases reported.

Dr. Stanton said he had seen a patient recover who had been treated with antistreptococcal serum at the Melbourne Hospital.

The seventh was the brain of a child, aged three and a half years. There was a history of vomiting and staggering gait which had developed some weeks before death.

The specimen contained a pedunculated tumour growing from the base of the brain, probably arising from the cerebellum.

The last specimen was a cerebral tumour, fungating in nature, which was situated on the lateral aspect of the left lateral ventricle.

PUBLIC MEDICAL OFFICERS' ASSOCIATION OF NEW SOUTH WALES.

THE third annual meeting of the Public Medical Officers' Association of New South Wales was held at the B.M.A. Building, 30 to 34, Elizabeth Street, Sydney, on January 21, 1929, DR. S. EVAN JONES, the President, in the chair.

Annual Report.

The annual report for 1928 was received and adopted. The report is as follows:

IN submitting its annual report for 1928 the Committee is pleased to record a busy year. Many schemes have been initiated, some prosecuted and a few brought to a satisfactory issue. It is necessary to remind our more ardent members that only by patient labours over many years will some of our objects be attained.

The Committee met fourteen times during the year, the number of attendances of individual members being as follow:

Dr. S. Evan Jones (President)	14
Dr. J. S. Purdy	12
Dr. H. H. Nowland	8
Dr. A. E. Machin	9
Dr. M. R. Finlayson	11
Dr. E. Sydney Morris	10
Dr. E. L. Morgan (Honorary Treasurer)	11
Dr. G. B. R. Wooster	6
Dr. Edelsten Pope	5
Dr. H. Hastings Willis (Honorary Secretary)	14

The number of members on the roll of the Public Medical Officers' Association now is eighty-three and should be larger. There are still some public medical officers who have not joined, but many of the junior medical officers are apparently as yet undecided whether they will remain in the services and so have not been requested to join us.

A public medical officers' association has been formed in Victoria during the year. The Committee has taken a lively interest in the formation of this association and hopes to collaborate with it in many matters of mutual interest in the future. The formation of similar associations in the other Australian States is considered desirable.

A proposal has been put forward that a conference of delegates from public medical officers' associations or similar bodies in Australia should be held in Sydney in 1929 at the time of the Medical Congress. The committee commends the proposal and submits it for the consideration of members.

Our relations with other professional bodies remain cordial.

Early in the year a proposal was made to us by the Society of Medical Officers of Health of Great Britain that this association should become a branch of that society. As the scheme submitted was financially disadvantageous to us and would possibly have involved an entire reorganization of our constitution with few and doubtful advantages to members, the committee viewed it with disfavour and has deferred consideration of our relationship to that body to a future date when the opinions of our colleagues in other States will be available.

A request was made to the New South Wales Branch of the British Medical Association for direct representation on the Branch Council. We have been informed in reply that an alteration in the constitution would be necessary and this the present council does not favour. As, however, there is in the local medical associations in New South Wales a strong feeling in favour of such an alteration, representations will be renewed as a favourable opportunity presents itself.

An opportunity was given the association to bring its activities under the notice of country delegates to the annual conference of local medical associations in Sydney on October 5, 1928. Our deputation received a sympathetic hearing.

With a view to keeping members informed of the association's activities, six circular letters have been distributed during the year. It is recommended that this practice be continued.

As instructed by the last annual general meeting, the Committee has considered the question of medical ethics and publicity. There seems to be a wide divergence of opinion on this matter and the Committee has been unable to arrive at a formula likely to receive sufficiently general support to be capable of enforcement. It is recommended that this matter stand over until public opinion in the association has crystallized more definitely.

The association, on June 21, 1928, gave a dinner in honour of Dr. Victor Heiser, of the Rockefeller Foundation, who was visiting Sydney. Thirty-five members were present and the function was a complete success.

The agitation for the establishment of post-graduate courses of medical study in Sydney has been continued. The Faculty of Medicine of Sydney University, the New South Wales Minister of Health and the New South Wales Branch of the British Medical Association have been approached on the subject, but so far no results have been obtained.

The methods of choosing Rockefeller scholars and the representatives of Australia at medical conferences abroad are considered unsatisfactory. Early in the year the Committee conveyed its views on these matters to the Federal Health Council suggesting that that body should nominate suitable persons to the Federal Government when an appointment is to be made.

Delegates were sent to the Federal Public Works Committee in April, 1928, to give evidence on behalf of the association in favour of the establishment of a school of tropical medicine and hygiene at the University of Sydney. It is noted that the Federal Government's scheme has been approved by Parliament. The arrangements for the control of the school contain some features which in the opinion of the Committee are undesirable, and the association may need to interest itself further in this matter.

The Committee's main task during the year has been its negotiations with the New South Wales Public Service

Board on the question of salaries. Full particulars of the results have been communicated to members in circular letters and recapitulation here is unnecessary. While far short of the association's demands, the board's concessions are a welcome advance upon existing rates and further benefits are possible when the subject again comes up for consideration in 1930. Certain minor salary increases have been granted in the services not under the New South Wales Public Service Board. These matters will call for further action in the New Year.

The objectionable practice of placing medical services under lay control will need to be strenuously opposed by this association. During the year the Brisbane City Council abolished its office of medical officer of health and placed its health department under a layman and a bill was introduced into the State Parliament of Victoria which would have united all the public medical services in that State into one department under a layman. The Committee has made representations to the Brisbane City Council, to the Federal Committee of the British Medical Association and to the New South Wales Branch of the British Medical Association on this matter and has drawn up a formula which it recommends for acceptance as the policy of the association, namely:

> The head of a medical service should be a medical practitioner. When that service is a department, the medical head should be the permanent head within the meaning of the *Public Service Act* and be directly responsible to the Minister and when the medical service is a branch of a department controlled by a board its head should be a member of that board and have direct access to the responsible Minister of the Crown.

The question of superannuation has received attention. Owing to a divergence of opinion among members, further instructions from a general meeting of the association are desirable.

Study leave and the recruiting of public medical officers are subjects which have received consideration during the year and may call for action by the incoming Committee.

A statement of the Association's finances is appended and it is hoped it will be considered satisfactory. After consideration the Committee does not recommend any reduction in the present rate of the annual subscription.

The Honorary Treasurer presented the financial statement for the year 1928. A credit balance of £32 1s. 6d. in current account and a reserve fund of £100 6s. 8d. were shown.

Election of Office-Bearers.

The result of the election of office-bearers for the ensuing year was as follows:

President: Dr. E. S. Stokes.
Honorary Secretary: Dr. H. Hastings Willis.
Honorary Auditor: Dr. J. A. L. Wallace.
Honorary Treasurer: Dr. E. L. Morgan.
Members of the Committee: Dr. M. R. Finlayson, Dr. S. Evan Jones, Dr. A. E. Machin, Dr. Dorothy McClemens, Dr. E. Morgan, Dr. H. H. Nowland, Dr. J. S. Purdy, Dr. C. J. M. Nowland, Dr. H. H. Willis.

Superannuation.

It was resolved to rescind a resolution passed at a special meeting in July, 1928, concerning superannuation, as it was reported that action called for by that resolution would be inexpedient.

Conference of Delegates.

On the motion of Dr. Willis, seconded by Dr. Harvey Sutton, the incoming Committee was authorized to arrange for a conference of delegates of public medical officers' associations or similar organizations in the several Australian States to be held in Sydney at the time of the third session of the Australasian Medical Congress (British Medical Association), in September, 1929.

Legal Defence of Members.

The question of the desirability of providing a system of legal defence of members who might be attacked in the courts of law or in Parliament as a result of the performance of duties of public medical officers was discussed. The Committee was asked to consider this matter.

Increments in Salaries.

It was decided that further representations should be made to the New South Wales Government concerning the delay in payment of the increments of salaries provided for by the agreement between the Public Medical Officers' Association of New South Wales and the Public Service Board.

Prizes for Medical Students.

DR. HARVEY SUTTON suggested that the interest on the vested funds of the Association should be used to provide prizes for medical students at the University of Sydney in preventive medicine and psychological medicine. Dr. E. Sydney Norris and Dr. J. A. L. Wallace supported the proposal which was referred to the Committee for consideration.

Obituary.

GABRIEL WILLIAM STAHEL FARMER.

GARRIEL WILLIAM STAHEL FARMER was born on January 11, 1865, at Sudbury in Middlesex, on the banks of the Thames. His father was John Farmer, a composer and organist. A few years later John Farmer was appointed teacher of music and organist at Harrow School. In due course Gabriel went to school at Harrow. He was a successful scholar and gained many prizes in classics, divinity and science. After he left Harrow he entered University College for the purpose of studying medicine. He did well at the medical school and in the early stages of his course he gained several certificates for efficiency. While he was still a student his father accepted a position at Balliol College, Oxford University, and the whole family moved to Oxford. Gabriel William Stahel Farmer continued his medical studies at Oxford and also took the arts course. He gained his B.A. before graduating in medicine. He subsequently took his M.A. degree. He was a hard working and successful student both at University College and at Oxford. After graduation he served at the London Hospital as House Surgeon and House Physician. In 1890 he graduated as M.B., B.Ch., at Oxford and passed the examinations for the diplomas of M.R.C.S., England, L.R.C.P., London. In 1892 he served as House Surgeon at the Royal Free Hospital in Gray's Inn Road. He then returned to Oxford. In 1894 he was appointed lecturer and demonstrator in anatomy. In the same year he secured the Fellowship of the Royal College of Surgeons of England and in the following year the degree of Master of Surgery at Oxford. He was then appointed examiner in anatomy. Shortly after this he gained a travelling scholarship for six months, the Radcliffe Fellowship. He visited many continental cities and worked under some eminent teachers. We are informed that an offer was made to him to extend his travelling research work for a further period of three years. He preferred, however, to take up private practice in Oxford. He retained his position as demonstrator of anatomy. In 1898 he was appointed junior honorary surgeon at the celebrated Radcliffe Infirmary, where he served for ten years. The work at the hospital and in his private practice was strenuous and heavy and in 1908 he had a serious nervous breakdown. For twelve months he was compelled to take complete rest without doing any work. He was then advised to take a voyage round the world. He joined the training ship *Port Jackson* and made two voyages in her. He made arrangements with the captain of the ship to give lectures to the cadets on history, geography, first aid and navigation. At the end of the second voyage in 1910 he decided to settle in Australia and he selected the township of Texas in Queensland. He was joined by his wife, to whom he had been married in 1895.

He soon gathered together a good practice and worked hard in the interests of his patients. In October, 1915, he joined the Australian Military Forces and was detailed to Enoggera Camp where he worked well for a time. In the following year he left Texas and settled in Maryborough. He practised in this town until the time of his death on February 1, 1929. He made many friends among all classes and his practice was a valuable one. During the last two years of his life Gabriel William Stahel Farmer suffered ill health in a patient and uncomplaining manner. Notwithstanding his disabilities, he continued with his work to the day of his death.

He was a kind and sympathetic companion and was ever ready to help his colleagues and friends in any way. His ability as a practitioner was considerable and he possessed much knowledge and skill in surgery. He leaves a widow and one brother and two sisters to mourn his loss.

JOSEPH VINCENT HIGGINS.

IN our issue of December 15, 1928, we announced the death of Joseph Vincent Higgins which occurred at Saint Vincent's Hospital, Melbourne. He was undergoing a small operation under general anæsthesia when one of those unavoidable accidents occurred that create so profound an impression on account of the unforeseen and tragic end.

Joseph Vincent Higgins was born at Swan Hill, Victoria, in 1900. He was educated at Saint Patrick's College, Ballarat, where he gained popularity among his fellows and the admiration of his teachers. He entered the Medical School of the University of Melbourne in 1918 and went into residence at Newman College. He was eminently successful as a student both in his work and at play. After having taken his double degree of bachelor of medicine and bachelor of surgery in 1925 he was appointed resident medical officer at Saint Vincent's Hospital, Melbourne, and at the Homeopathic Hospital in Melbourne. Later he obtained the position of resident medical officer at the Saint Vincent's Hospital, Toowoomba, Queensland. After having completed this service, he started private practice in Johnston Street, Fitzroy. He was well beloved by his fellows and was known for the generosity of his actions among the poor of the industrial suburb in which he practised. He was a young man of promise and by his sudden and unexpected death we are left the poorer.

Research.

SCIENCE SCHOLARSHIPS AND GRANTS.

THE SCIENCE COMMITTEE OF THE BRITISH MEDICAL ASSOCIATION has issued the following notice to which we desire to direct the attention of members. It will be remembered that arrangements have been made whereby the Federal Committee of the British Medical Association in Australia will act on behalf of the Council of the Association in connexion with the scholarships and grants in aid of scientific research.

Scholarships.

The Council of the British Medical Association is prepared to receive applications for research scholarships as follows:

An Ernest Hart Memorial Scholarship of the value of £200 *per annum;*

Three research scholarships, each of the value of £150 *per annum.*

These scholarships are given to candidates whom the Science Committee of the Association recommends as qualified to undertake research in any subject (including State medicine) relating to the causation, prevention or treatment of disease. Each scholarship is tenable for one year, commencing on October 1, 1929. A scholar may be reappointed for not more than two additional terms. A scholar is not necessarily required to devote the whole of his or her time to the work of research and may hold a junior appointment at a university medical school or hospital, provided the duties of such appointment do not interfere with his work as a scholar.

Grants.

The Council of the British Medical Association is also prepared to receive applications for grants for the assistance of research into the causation, treatment or prevention of disease. Preference will be given, other things being equal, to members of the medical profession and to applicants who propose as subjects of investigation problems directly related to practical medicine.

Conditions of Award: Applications.

A copy of the regulations relative to the award of the scholarships and grants in 1929 and of the prescribed application form can be obtained on application to Dr. R. H. Todd, Secretary of the Federal Committee of the British Medical Association in Australia, B.M.A. Building, 30 to 34, Elizabeth Street, Sydney. The completed application form is required to be submitted to Dr. R. H. Todd not later than March 16, 1929. Applicants are required to furnish the names of three referees who are competent to speak as to their capacity for the research contemplated, to whom reference may be made.

ALFRED COX,
Medical Secretary.

British Medical Association House,
Tavistock Square, London, W.C.1,
November, 1928.

New Year Honours.

HIS MAJESTY THE KING has been pleased to confer the following honours on medical practitioners. The announcement of the lists may be regarded as an indication of His Majesty's satisfactory progress towards recovery.

Sir Berkeley Moynihan, President of the Royal College of Surgeons of England, has been elevated to the peerage.

Dr. Norman W. Kater, M.L.C. (New South Wales) has been created a knight. It is announced that the honour is in recognition of his services as chairman of the Federal Pastoral Advisory Committee. Sir Norman Kater has not practised his profession of medicine for many years, but has attained much prominence in political and commercial life.

Dr. Carrick H. Robertson, of Auckland, has also received the honour of knighthood. His eminent position in surgery is too well known to require elaboration in this place.

Dr. J. H. L. Cumpston, Director-General of Health of the Commonwealth, has been created a Commander of the Order of Saint Michael and Saint George. His distinguished services in the realm of hygiene and preventive medicine are thus receiving public recognition.

Professor Anne McIlroy, whose important work as honorary obstetrician at the Royal Free Hospital has placed her in the front rank of British obstetricians, has been made a Dame Commander of the Order of the British Empire. Professor Winifred Cullis, Professor of Physiology at the London School of Medicine for Women, has earned the same distinction. The Companionship of Honour of the Order of the British Empire has been bestowed on Lady Barrett, Dean of the London School of Medicine for Women.

But few medical practitioners have been made peers in recognition of their professional and scientific attainments. Because of rarity of this event it is particularly gratifying that the President of the Royal College of Surgeons has received this honour.

The congratulations of the medical profession in Australia are extended to all the recipients of honours and more especially to Sir Carrick Robertson, Dr. J. H. L. Cumpston and Sir Norman Kater.

Books Received.

VARICOSE VEINS AND THEIR TREATMENT BY "EMPTY VEIN" INJECTION, by Ronald Thornhill, M.B., Ch.B., with Foreword by Sir Berkeley Moynihan, Bart., K.C.M.G., C.B., M.S., F.R.C.S.; 1929. London: Baillière, Tindall and Cox. Crown 8vo., pp. 74. Price: 5s. net.

CLINICAL OBSERVATIONS ON INFANT FEEDING AND NUTRITION, by Howard B. Gladstone, M.D. (Edinburgh); 1928. London: William Heinemann (Medical Books) Limited. Demy 8vo., pp. 130. Price: 7s. 6d. net.

PRACTICAL CHIROPODY, by E. G. V. Runting, F.I.S.Ch.; 1928. London: Faber and Gwyer, Limited. Crown 8vo., pp. 210, with illustrations. Price: 5s. net.

PHYSIO-THERAPY IN GENERAL PRACTICE AND FOR THE USE OF MASSEUSES, by E. Bellis Clayton, M.B., B.Ch. (Cantab.); Second Edition; 1928. London: Baillière, Tindall and Cox. Royal 8vo., pp. 241, with illustrations. Price: 12s. 6d. net.

EXPERIMENTS WITH HANDWRITING, by Robert Saudek; 1928. London: George Allen and Unwin, Limited. Royal 8vo., pp. 393. Price: 18s. net.

DISEASES OF CHILDREN, Edited by Hugh Thursfield, D.M. (Oxon.), M.A., F.R.C.P., and Donald Paterson, M.B. (Edinburgh), M.R.C.P.; Second Edition; 1929. London: Edward Arnold and Company. Royal 8vo., pp. 1117, with illustrations. Price: 45s. net.

Diary for the Month.

MAR. 12.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAR. 12.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAR. 14.—Victorian Branch, B.M.A.: Council.
MAR. 14.—New South Wales Branch, B.M.A.: Meeting of the Section for the Study of Cancer.
MAR. 19.—New South Wales Branch, B.M.A.: Council.
MAR. 20.—Western Australian Branch, B.M.A.: Branch.
MAR. 21.—New South Wales Branch, B.M.A.: Branch.
MAR. 22.—Queensland Branch, B.M.A.: Council.
MAR. 26.—New South Wales Branch, B.M.A.: Council.
MAR. 27.—Victorian Branch, B.M.A.: Council.
MAR. 28.—South Australian Branch, B.M.A.: Branch.

Medical Appointments.

Dr. John Coffey (B.M.A.) has been appointed Commissioner of Public Health and Inspector, School of Anatomy, Brisbane, Queensland.

* * *

Dr. Harry Roy Pomroy (B.M.A.) has been appointed Acting Medical Officer, Venereal Clinic (Male Section) at the Adelaide Hospital.

* * *

Dr. Gilbert Edgar Jose (B.M.A.) has been appointed Acting Honorary Clinical Assistant to the Venereal Clinic (Male Section) at the Adelaide Hospital.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

AUSTIN HOSPITAL FOR CHRONIC DISEASES, HEIDELBERG, VICTORIA: Junior Resident Medical Officer (male).
DEPARTMENT OF INSPECTOR-GENERAL OF HOSPITALS: Junior Medical Graduates (four).
HAMILTON AND DISTRICT HOSPITAL, VICTORIA: Resident Medical Officer.
JANEFIELD SANATORIUM, BUNDOORA, VICTORIA: Resident Medical Officer.
ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Honorary Consulting Urologist.
THE BRISBANE AND SOUTH COAST HOSPITALS BOARD: Medical Vacancies.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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